PSYCHO-PHYSIOLOGICAL REACTIVITY AND PERSONALITY IN BRONCHIAL ASTHMATICS

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SUMMARY

Chapter 1

The aims of the research were twofold; one was to investigate the personality characteristics of a group of bronchial asthmatics, and the other was to examine the relationships between data gathered by the two methods of assessment used; psychological questionnaire measurements of personality and psychophysiological measurements. It was argued that psychophysiological assessment was especially suitable for the investigation of asthma with its respiratory dysfunction due to the nature of the neural mechanisms controlling respiration, and because recent advances in the assessment of anxiety were based on psychophysiological techniques. The specific points under investigation were -

a. Are the predominant personality traits observed in a group of asthmatics different from those found in comparable control groups of normal and anxious subjects?

b. Do asthmatics differ from these two groups in their psychophysiological response to stress, either quantitatively (in the magnitude of their responses) or qualitatively (by displaying different patterns of response compared with the control groups).

c. Can the use of psychophysiological techniques previously used for the assessment of anxiety in a group of anxious patients

be extended to assess anxiety in asthmatics?

d. Are there any consistent relationships between personality and psychophysiological measures, and are these relationships the same for asthmatics and non-asthmatics?

Chapter 2

The review of literature covered the fields of psychological studies of asthma, studies of individual differences in psychophysiological function, and the theoretical and experimental approaches to the investigation of the relationship of psychophysiological variables and personality.

Chapter 3

This chapter described the subjects and the sampling methods by which they were obtained. Four groups of subjects, randomly selected asthmatics, asthmatics referred to psychiatrists, and normal and neurotic controls were studied. The procedure followed in testing the subjects was described.

Chapter 4

Two "objective" techniques measuring indicants of anxiety were described - habituation of the psychogalvanic reflex, and measurement of forearm blood flow by venous occlusion plethysmography. The literature relevant to each of these techniques was reviewed and the procedures outlined. No significant differences were found between the normal and neurotic groups on any of the measurements obtained. It was concluded that since no discrimination between two groups deliberately chosen to be bipolar on the dimension of

anxiety had been obtained, the techniques could not be used in this study for assessment of the asthmatics. Some possible reasons for the negative results were discussed.

Chapter 5

The four tests used, the Eysenck Personality Inventory, the Taylor Manifest Anxiety Scale, the Hostility and Direction of Hostility Questionnaire and Cattell's 16 Personality Factor Questionnaire were briefly described. These tests showed that the asthmatics lay between the normal and neurotic groups on scales measuring anxiety, hostility and neuroticism. When compared with normative data obtained from the general population, the asthmatics were significantly more neurotic, submissive, and tough minded.

Chapter 6

The methods used to produce an external airway resistance and to record the psychophysiological responses to the stressor were fully described. The results of this experiment indicated that there were no differences between asthmatic and normal subjects in the magnitude of their physiological responses to the airway resistance. Estimates of the reliability of response and basal level measures showed the latter to be more reproducible, both within and between occasions of testing. An analysis of individual response patterns showed that there was a slight tendency for the asthmatic group to show evidence of greater response and basal activity in respiratory rather than non-respiratory measures.

A factor analysis of the psychophysiological and psychological data showed that in the asthmatic group only, high anxiety was linked with increased respiratory responsiveness. A comparison of individuals who showed specificity of basal level response patterns with those who did not indicate that the latter might be more anxious.

Chapter 7

The results of the study were summarised, and several factors which might have contributed to the results obtained were examined. The results obtained were not incompatible with previous work and indicated that future studies of asthmatics could usefully be performed. They should employ a variety of stressors, and attempt in addition to examine cognitive and subjective responses to them. The implications of the findings for the clinical management of asthmatics were pointed out. It was concluded that psychophysiological measurements were best considered in the context of the psychological processes occuring contiguously. Theoretical approaches which did not assume a parallelism of physiological and psychological function, but assigned a separate biological role to measurements of physiological arousal were considered desirable to guide future research.

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The work incorporated in this dissertation was carried out in collaboration with Dr. R.C.B. Aitken and Dr. A.K. Zealley, and formed part of the larger project. This also included psychiatric interview assessments of the individuals studied, with which I was not concerned. My active participation in the research consisted of the administration of the psychological tests, and overall responsibility for all of the psychophysiological studies. The analysis of the data so obtained and the conclusion drawn from them are my complete responsibility. Such matters as the selection of the subjects and the overall strategy of the study were necessarily joint decisions, taken by all three individuals involved in the research.

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Preliminary reports of some of the results incorporated in this thesis have already been published. Copies of these papers may be found in Appendix 5.

CONTENTS

CHAPTER 1

INTRODUCTION AND AIMS OF THE STUDY

"In this world, things are complicated and are decided by many factors. We should look at problems from different aspects, not from just one".

-- Quotations from Chairman Mao Tse-Tung

It has been said that psychologists bear an uncanny resemblance to the blind men who were trying to describe an elephant. These wise gentlemen, it will be recalled, vehemently disagreed over what was the true nature of the beast, since they each had hold of different parts, and could not bring their separate impressions together.

To guard against obtaining such a limited view of the subject matter of this thesis, two types of measurement techniques have been used. These were psychophysiological and psychometric measures. The principal aim of the research reported here was to investigate the psychological characteristics of a group of asthmatics; specifically, did they differ from normal and anxious control groups on a variety of psychophysiological and psychological tests? But in order to take the vantage point of the man who could both see and feel the elephant, it is also important to be aware of the relationships between the two types of observation. Thus a second aim of the research was to enquire into the relationships between differences in psychophysiological functioning and personality traits.

The investigation of the psychological characteristics of a group of asthmatics seemed a particularly appropriate field of inquiry for the combined use of psychological and psychophysiological observations. The principal symptom of the disorder - the sensation of breathlessness - is one in which psychological and physiological processes are both implicated, especially in the situation where the patient is attempting to communicate his feelings of distress arising from his breathlessness.

It is worth considering how the bronchioconstriction which characterises asthma leads to breathlessness. The initial effect is to increase the difficulty of breathing, since the cross-sectional area of the bronchioles is decreased. Thus greater mechanical work will need to be performed to move the same volume of air in and out of the lungs, from a simple consideration of Poiseuille's Law. This hindrance of breathing has been put forward by Campbell and Howell (1963) as one of the conditions under which the sensation of breathlessness is experienced. Their theory suggested that a factor that they called "length: tension inappropriateness" was responsible for the neural signals to the reticular formation in the medulla which indicated the demand for a change in respiratory function. This concept of length:tension inappropriateness refers to the ratio of the demand for ventilation and the muscular tension (or effort) needed, which is thrown out of balance in a situation of breathlessness. Their theory will not be elaborated in detail, but it is easy to see that the increased muscular effort resulting from broncho-constriction would set

up a situation of inappropriateness, and give rise to neural activity indicating breathlessness. Were this breathlessness to be followed by enhanced ventilation, the increased air flow necessary to sustain this would also be a cause of increased mechanical work, and an already disturbed situation could thus be aggravated further.

The psychological importance of this theory stems from a consideration of the various neural structures and mechanisms involved in the complex servo-system controlling breathing. The reticular formation, which receive the afferent signals, cause compensating changes in both the α- and γ- muscular systems, and the diffuse nature of the γ system may lead to increased activity being found outside the respiratory muscles, causing general muscle tension, a sign of anxiety. In addition, neural pathways are known through which signals from the peripheral nervous system are relayed to the limbic system, where the emotional state of the individual is modulated (Maclean, 1955).

Conversely, emotional arousal could aslo cause generalised muscle activity, extending to the respiratory musculature, which could thus be a direct cause of dyspnoea. Whether one espouses James and Lange's view that peripheral physiological changes are the basis of the experience of emotion, or the opposing views of Cannon, there are ample grounds for surmising that the phenomena may be intimately related, on physiological grounds alone.

Over and above physiological considerations are more psychological ones. Breathing is so critical to the maintenance of life, that when dyspnoea occurs it may well be perceived as a direct threat to continued existence. Were worsening dyspnoea to reach asphyxia, it would be fatal, and the experience of acute anxiety is thus a not unlikely consequence of breathlessness. This appraisal of threat will be determined in part by individual personality traits, as well as by objective changes in respiratory function. Comroe (1966) has emphasised that breathlessness, the sensation of dyspnoea, is subjective; and such varied factors as the ability to perceive small changes in sensation, as well as the influence of specific personality traits in their appraisal, will all contribute to the total reaction.

There are many questions without satisfactory answers about the personality characteristics of asthmatics. Would the traits commonly found in a group of these individuals cause them to be oversensitive to the occurrence of dyspnoea, and to experience severe anxiety at the onset of an attack of asthma, with its uncertain duration and severity? Many of the assertions on this subject have been little more than speculation, and most workers have been preoccupied with problems of the aetiology of bronchospasm. Additionally, (this will be discussed in detail subsequently), many studies of the personality of asthmatics have been deficient due to the highly unrepresentative nature of the groups of individuals studied. Thus there appeared good reason to undertake the assessment of personality in a large and representative group of asthmatics.

Since the trait of most interest was clearly anxiety, it was decided to compare the asthmatics with two control groups bipolar on this dimension. Thus the investigation of groups of normal and neurotic subjects was also incorporated in the study.

Psychophysiological techniques are, of course, particularly appropriate for the study of anxiety. In the last few years, "objective" psychophysiological techniques have emerged for the assessment of anxiety in a clinical setting. One of the aims of this research was to investigate the use of these techniques -- habituation of the psychogalvanic response and forearm blood flow -- in assaying anxiety in a non-psychiatric, but possibly overall quite anxious group of asthmatics. Previously, these two techniques had only been used in assessing anxiety in known anxiety-prone groups of psychiatric patients, and success in using them to assess anxiety in the asthmatics would be a welcome additional demonstration of their usefulness.

Another use of psychophysiological measures is in assessing the response to psychological stress, and a further aim of this research was to see if the asthmatics' response to stress differed in any way from anxious and non-anxious control groups. The stressor used was an external airway resistance which made expiration difficult while it was applied. This was chosen to be deliberately meaningful for the asthmatic group, since it slightly resembled the difficulty in expiration that is encountered during an attack of asthma.

It should be made clear at this point that the author's interpretation of the data obtained will be primarily a psychological one. Although the pathophysiology of breathlessness, reviewed earlier, was constantly borne in mind, it was felt that the magnitude of the external airways resistance used as a stressor was so small that its impact was primarily symbolic and psychological. None of the asthmatics developed severe bronchospasm, and there is no evidence that any of the subjects were unduly disturbed by the experimental treatment.

Two main questions were of interest. Did the magnitude of the asthmatics' physiological responses to this stressor differ from that of the other two groups of subjects; and would the patterning of the asthmatics' responses differ from that of the other two groups?

There is evidence, to be reviewed later, that differences in physiological responsivity are in some way associated with different forms of emotional expression, and in general, to individual differences in personality. Thus a further series of questions was posed: namely, were individual differences in physiological functioning stable enough to characterise the individual, and were these differences (both qualitative and quantitative) linked to other psychological measures? Few workers have paid much attention to the stability of physiological measures, while at the same time there have been claims that they are linked to stable personality traits; therefore, an investigation of stability was indicated and was carried out. Finally, a most intriguing question was whether these psychophysiological inter-relationships were the same for asthmatic and non-asthmatic groups.

There will be little attempt to discuss why these two levels of human functioning, the psychological and psychophysiological, should be linked together. It is the writer's view that such a complex question can best be investigated within a developmental framework, where the full relationship between constitutional and environmental influences on personality development can be examined over a long period of time. To speculate on it with insufficient data would be to fall prey to another regrettable habit of psychologists -- premature use of the armchair and its theories as a tool of enquiry. The first task of a natural science -- particularly one dealing with the complexities of human beings -- is systematic observation. Only after this can the cycle of hypothesis, theory and experiment follow. It is this initial stage of observation, backed by the power of such descriptive statistical techniques as factor analysis, which is reported in the next six chapters.

CHAPTER 2

REVIEW OF LITERATURE

Introduction

The research conducted for this thesis studied the psychological and physiological characteristics of asthmatics and non-asthmatics and the interrlationships between these measures. Accordingly, this review is divided into three sections, each dealing with one aspect. These are followed by a summary which attempts to integrate the main findings of the preceding three sections: these sections comprise -

A. A review of psychological studies of asthmatics.

B. A review of psychophysiological studies of asthmatics.

C. A review of studies on the nature of individual psychophysiological differences.

D. A review of work relating personality differences to psychophysiological ones.

A. Psychological Studies of Asthmatics

1. Theoretical approaches

> "The term bronchial asthma ... is employed to describe recurrent, generalised airways obstruction, which at least in the early stages is paroxysmal and reversible ... The most important manifestations are dyspnoea and wheeze"
> (Crofton & Douglas, 1968).

From early times, the aetiology of this disease has been associated with psychological factors. Hippocrates cautioned the asthmatic to 'guard himself against anger', and more recently, Henry Hyde Salter (1860) put forward the opinion that asthma was but 'a particular form of perverted nervous action'. However, it was not until many years later that this approach was expanded by Franz Alexander. His influential theory of psychosomatic disease (1950), although partly based on untestable psychoanalytic concepts, is of note simply because it was the first systematic attempt to relate psychological and physiological phenomena is an understanding of the disorder. The theory postulates the presence of three factors in the genesis of psychosomatic disease. A person with a particular organic pre-disposition (or organ vulnerability) and an emotional conflict pattern characteristic of a specific psychosomatic illness, will develop the illness only when specific emotional situations relating to his central conflict arise and mobilize it. This arousal leads

to a breakdown of his psychological defenses, which in turn precipitates (or exacerbates) his somatic symptoms. In other words, asthma is a substitute response which serves the psychological needs of the individual.

In the asthmatic the respiratory system is the vulnerably predisposed organ and the conflict is the fear of separation from a loved person (usually the mother). However, most of the evidence for Alexander's theory has come from individual case studies rather than controlled investigations, and in a recent review of studies of the mother-child relationship in asthma, Kelly & Zeller (1969) conclude that "no conclusion can be drawn at this time as to the nature and significance of the family factor in asthma".

Other workers have tried to interpret asthma attacks as a learned response, and Turnbull (1962) has summarized the implications of a learning theory approach to the disease. Various studies have been reported in which 'learned asthma' could be induced both in infrahuman and human subjects. Dekker & Groen (1956) showed that certain asthmatics would react with an 'attack' of asthma when they were placed in a laboratory imitation of what they had previously described to be an 'asthmatogenic situation'. They noted that high intensity of emotion <u>per se</u> was not sufficient to cause an attack, but the nature of the emotion was important, an observation in line with Alexander's theory. Ottenberg & Stein (1958) showed that conditioning could produce asthmatic attacks in guinea pigs. Animals placed in a cage in which they had previously received sensitizing injections of egg

white, causing an 'asthma' attack, developed these attacks without the injection being given. However, Franks and Leigh (1959) investigated eyelid conditioning in asthmatics, neurotics, and normals, on the basis of the Eysenckian personality theory prediction that individual differences in introversion-extraversion are linked with ease of conditioning. No differences between any of the groups or any of the measures was found, nor did the expected negative association between extraversion and conditioning emerge. Purcell (1965) reviewed studies on "conditioned" asthma and concluded that several of the studies claiming to show this phenomenon just did not do so -- they were able to show respiratory dysfunction resulting from a conditioning situation, but there was rarely evidence that asthma was produced. It is safe to conclude from the work reviewed here that while learning (in the broadest sense) may be important in many asthmatics, the conditioning approach by itself has little heuristic value.

2. Descriptive Studies of Psychological Factors in Asthma

There has been a wealth of investigations into the psychological characteristics of the asthmatic, which have been reviewed by Leigh (1953), Freeman, et al. (1964), Kelly & Zeller (op. cit.). However, many of these studies have been carried out on highly selected samples of asthmatics -- often ones with concurrent psychiatric disorder -- and there has been a tendency for the results to be generalised to asthmatics as a whole. Leigh and Marley (1956) found that asthmatic patients attending a psychiatric hospital showed more signs of depression, anxiety, sensitivity and anger when assessed on the Cornell Medical

Index (CMI) than a comparable 'non-psychiatric' group attending a general hospital. The 'psychiatric' asthmatics had similar CMI profiles to a control group of neurotic patients. Thus one cannot generalise any findings from a 'psychiatric' sample to other groups of asthmatics. However, even taking a sample of asthmatics who attend a respiratory diseases clinic can lead to false conclusions as to the role of psychological factors in the disorder.

Zealley (1971) compared a sample of 12 women who had attended a chest clinic on one particular day with a randomly chosen sample from the register of asthmatics at the same clinic. The two samples were similar in age, time since the first attack of asthma, and duration of chronic asthma. He retested the 'attenders' two years later, using the Eysenck Personality Inventory (EPI) (Eysenck & Eysenck, 1964) and the Hostility and Direction of Hostility Questionnaire (Caine, Foulds & Hope, 1967). The 'clinic attenders' had high Neuroticism scores on the EPI when first tested, and two years later their Total Hostility Score on the HDHQ had decreased (notably in the subscale "Criticism of Others"), though their Neuroticism scores had not changed. The group of asthmatics chosen at random had normal scores for both Neuroticism and Total Hostility. Besides being a timely warning that the sampling procedure in studies of this sort can radically influence the results, this study also shows that being an asthmatic is not necessarily associated with neuroticism and other psychopathology <u>per se</u>, but that other factors (the author suggests severity of asthma, and the psychic support obtained at the clinic) also contribute to the picture.

Oswald *et al.* (1970) claimed that out-patient asthmatics and bronchitics showed increased scores on measures of neuroticism, anxiety and introversion compared with the general population, and that neuroticism and anxiety scores were correlated with increased respiratory disability.

Studies using unselected groups of patients have not confirmed the existence of a specific 'asthmatic' personality. Rees (1956) assessed personality factors in 441 asthmatics attending an allergy clinic and a group of control subjects. He found that the asthmatics showed a significantly higher incidence of such personality traits as anxiety, timidity, sensitiveness and obsessionality. While there was no one personality trait specifically associated with the asthmatics, he commented that all of these traits, in different ways, were conducive to the development of states of emotional tension which could act as precipitants of attacks of asthma. The possibility that these traits were a result of asthma rather than a possible cause of it was rejected. Rees felt that no single set of factors -- psychological, infective or allergic -- could be said to be the cause of the disease in most patients.

The study by Leigh & Marley (*op. cit.*) showed that on the basis of the CMI, asthmatics were more vulnerable, rigid, and less able to tolerate anxiety than a control group of normal subjects. In contrast, Franks and Leigh (1959) gave the Maudsley Personality Inventory to a group of asthmatics attending hospital as psychiatric out-patients, two groups of neurotics and a group of normal controls. The asthmatic patients occupied a position midway between the neurotics and normals on the Neuroticism scale, though not differing significantly from either group.

The authors concluded that the neuroticism may be reactive to the dysfunction itself, though they provided no evidence for this conclusion. Dekker, Barendregt and DeVries (1961) showed that there was no difference in Neuroticism scores on the Heron Two Part Personality Inventory between an "allergic" group of asthmatics (those in whom asthma could be provoked by inhaling nebulized allergens) and non-allergic ones. There was no difference in scores between the asthmatics and an age-matched group of neurotics, but the asthmatics scores were significantly higher than those of a control group of normal subjects.

Purcell et al. (1969) divided a large group of asthmatic children into subgroups on the basis of an Allergic Potential Scale (APS), the severity of their asthma, and a rating of the relevance of upper respiratory tract infection to asthma. Each individual group was also divided in terms of rapid remitters (children who had had little asthma since being institutionalised) and steroid dependent (those who needed almost continuous drug treatment). The personality measures used were the Children's Personality Questionnaire and the High School Personality Questionnaire. They found that there was greater evidence of psychopathology in the Low APS/Rapid Remitters group compared with the High APS/Rapid Remitters group, although the two groups were nearly identical in severity of asthma. A comparison of the attitudes of the parents of children in the Low APS/low relevance of infection group to those in the High APS/high relevance of infection group, revealed that the parents of the former group displayed more authoritarian and restrictive attitudes to their children.

Hirt et al. (1968) found no relationship between any Minnesota Multiphasic Personality Inventory (MMPI) scores and a rating of "allergic predisposition" to asthma. However, their sample of 30 patients was selected from the private practice of a single allergist and may not therefore have been a very representative sample.

Several studies have investigated the expression of hostility in asthmatics. Miller & Baruch (1950) suggested that asthmatic children showed aggression and hostility less than non-asthmatic controls but that such hostility as they did express was intropunitively directed. In contrast, Beech & Nace (1965) found that, using a sentence completion test, asthmatic children were able to express hostility more readily than a criterion group of "non-aggressive" psychiatric referrals, but did not differ from "aggressive" or "intermediate-aggressive" groups of children. However, the authors point out that the verbal expression of hostility is quite different from the ability to "act out". Pierloot and Van Roy (1969) gave the Rosenzweig Picture Frustration Test (a projective test) to 30 adult female asthmatics and comparable groups of psychoneurotic and healthy subjects. Asthmatics gave a larger number of impunitive (showing suppressed aggression) responses than the other two groups. They gave significantly fewer extrapunitive responses than the normal subjects. These results are generally in agreement with those of Miller and Baruch (op. cit.).

B. Psychophysiological Studies on Asthmatics

1. Theoretical Contributions

The respiratory tract is supplied by both the sympathetic and parasympathetic branches of the autonomic nervous system. The parasympathetic motor nerves supply the bronchoconstrictor muscle and the mucus glands of the bronchial mucosa. These can be stimulated reflexly by afferent parasympathetic nerves in the tract (by the presence of mucus or foreign matter in the bronchi), but also from impulses from higher nervous centres. In status asthmaticus, a vicious circle may well operate, whereby bronchospasm is mediated parasympathetically, which results in increased mucus secretion which in turn causes further parasympathetic reflex stimulation.

Sympathetic stimulation causes dilation of the bronchiolar muscle. This is why an injection of adrenaline (whose effects on the nervous system are similar to that of sympathetic stimulation) in status asthmaticus can bring relief, and break the vicious circle just referred to. Since the physiological changes associated with such emotions as fear and anxiety also involve sympathetic discharge and adrenaline secretion, it is at least possible that the nervous activity associated with emotional experience could interact in a complex fashion at a neural level with the physiological changes associated with an attack of asthma.

Rees (1965) reviewed the role of the autonomic nervous system in asthma. He noted that the functional disturbances in allergy involve changes in the musculature of the viscera and generally increase cholinergic activity. He explained why emotional tensions and allergic

reactions could have an additive effect on the functioning of autonomic nervous system. Rees felt that the integrating concepts of stress and homeostasis could be valuable in considering the interaction of different aetiological factors in asthma, but did not pursue this line of thought in any detail.

Robinson (1967) attempted to reinterpret Alexander's psychodynamic views on the role of hostility in asthma in terms of the physiological components of hostility and anxiety. Using the ideas of autonomic balance (Wenger, 1948, 1966) and autonomic tuning (Gellhorn and Loofbourrow, 1963) he has shown how the experience of prolonged hostility (an "onset situation") could lead in sensitive individuals to massive parasympathetic activity. This would occur as a reaction following the sympathetic activity associated with the emotion, and would probably be the immediate precursor of an attack of asthma. Unfortunately he does not present any evidence that these physiological changes in fact take place in asthma.

2. Experimental Contributions

Wenger (1948) reported determinations of autonomic balance in a group of 16 asthmatics. Compared with a normative group of 488 aviation students the following differences were found:

a) significantly lower dermographia persistence

b) lower log conductance response to stress

c) higher systolic and diastolic blood pressure

d) less sinus arrhythmia

e) higher heart rate

f) higher respiration rate

g) lower tidal volume

h) lower standard deviation of tidal volume

At the time, Wenger concluded that these differences did not support the idea that asthmatics displayed parasympathetic dominance, as most of them would be caused by increased sympathetic nervous system activity. Later, Wenger (1957) reinterpreted some of the data. It had been noted that originally only about one-third of the normal group tested showed patterns of sympathetic or parasympathetic dominance in a pure form. By looking at the modal pattern for the abnormal groups he found a mixed pattern of responses which he called "Alpha"; and a similar mixed one named "Beta", which was found in many individuals with psychosomatic disorder. The frequency of occurence of this pattern in "psychosomatic" patients and in other groups (neurotics and schizophrenics) differed significantly from that in the normative sample. However, in a retrospective study (Wenger, 1966) of the value of autonomic balance scores obtained 20 years previously as an aid in predicting psychosomatic and psychological disorder, few respondents reporting asthma over those 20 years had shown the "Beta" pattern when first tested. Wenger concluded rather wistfully that "there is a specific mixed pattern for men who developed asthma which we have not discovered ... Additional studies of asthmatic patients are again indicated".

Hahn (1966) compared the physiological responses of 20 asthmatic children and 10 non-asthmatic children in a problem-solving situation. He showed significantly higher heart rate and skin temperature among the asthmatics. There was no difference between the groups in the frequency of spontaneous skin conductance activity. This high incidence of tachycardia was confirmed by analysing electrocardiograms of a further 352 chronic asthmatic children. The regression equation of heart rate under stress on basal heart rate did not correspond with that of the normal children, and the author suggested that there may be a possible malfunction of homeostatic mechanisms in this group. In a further study (Hahn and Clark, 1967), 18 symptom-free asthmatic and 21 non-asthmatic children were given tone, shock, and problem-solving stimuli. When the children were criticised during problem-solving, the asthmatics reacted with greater negative affect, and, although showing equal or greater arousal than non-asthmatics, did not continue to show increases in respiration rate during the most stressful procedures. A difference between the asthmatics and normals in the regression of post-stimulus on pre-stimulus heart rate was also found in this study, with the asthmatics showing an increase in heart rate at high pre-stimulus levels in contrast to the normals, who showed the decrease that the Law of Initial Values would predict.

Selesnick et al. (1969) showed films to 28 adult asthmatics depicting the sort of emotional conflict situations that Alexander (op. cit.) would expect to trigger attacks of asthma, and monitored heart rate, respiration rate, finger temperature, neck temperature,

skin conductance and expiratory irregularities. The viewing took place a week after the subjects had been randomly assigned to treatment with an anxiety reducing drug, diazepam or a placebo. The diazepam group showed fewer expiratory irregularities in response to the stressor film than the placebo group, and a greater vital capacity. No differences were found in the other measures. They interpreted their finding that only the respiratory system showed differential reactivity to diazepam as evidence for the theory of organ specificity; however, without a comparable group of non-asthmatic subjects this conclusion is not tenable.

There have been other studies of the effect of 'emotional' situations on the respiratory system in asthmatics, but only one of these has employed suitable control groups. Ziegler & Elliot (1926), Treuting & Ripley (1948), Stevenson & Ripley (1950) have all shown that thinking about or discussing emotional events of relevance to a particular asthmatic subject could lead to respiratory changes. Doust and Leigh (1953) showed that emotional tension expressed in a psychiatric interview could lead to anoxaemia in asthmatics. In the one controlled study, Luparello et al. (1968), subjects were made to believe that they were inhaling irritants and allergens leading to bronchoconstriction (in fact they were breathing nebulized saline solution). Nine out of 40 asthmatics reacted with a significant increase in airways reactivity and 12 developed full-blown attacks of asthma, which could be reversed with a placebo inhaler (saline solution again!). None of the 40 control subjects reacted in this way.

There is ample evidence, however, that profound changes in breathing can be obtained in non-asthmatic subjects in association with emotional feelings. Indeed, the relationship has been claimed to be causal, for as early as 1840, John Perceval wrote that "well regulated breathing is essential to bodily health and mental restoration".

Rather more modest claims have been made by later workers. Nielsen and Roth (1929) maintained that certain types of spirogram were associated with neurotic and psychiatric disorder, and Sutherland et al. (1938) claimed that spirograms were characteristic of the individual. Dudley et al. (1964) investigated the effect of hypnotic suggestion of naturally occuring or experimentally induced psychological stress on the respiratory system. In their subjects (11 young men) they observed two patterns of response. The "action-oriented" pattern, which was produced in response to suggestions of anger, anxiety, and exercise, consisted of elevated alveolar ventilation and oxygen consumption, and decreased alveolar CO_2 concentration. The other pattern (non action-oriented) was produced in responses to suggestions of relaxation and depressive feelings, and was essentially the converse of the action-oriented pattern. They found that mixed emotional responses were accompanied by mixed physiological patterns.

The same group of workers (Dudley et al., 1967) have investigated the incidence of dyspnoea (the sensation of breathlessness) in both normal subjects and patients with pulmonary disease. This was found not to be related to any measures of ventilation, (minute volume, alveolar ventilation, respiration rate and tidal volume). In subjects with pulmonary disorders, dyspnoea occured as a result of physiological and psychological change. It was found to be associated with the hyperventilation and hyperpnoea related to anxiety, and also to the hypoventilation associated with depression. The authors felt that dyspnoea could be explained on an individual basis by past conditioning experiences such as the association of threatening or painful events with breath-holding, crying, and other respiratory behaviour. This explanation clearly has parallels in the conditioning approaches to asthma mentioned earlier. These experiences would subsequently interfere with the perception of signals from the cardiopulmonary system to give rise to the sensation of dyspnoea. It is possible that the anxiety evoked by the dyspnoea could increase the distress and create a vicious circle, although in dyspnoea caused by asthma the anxiety (if it is accompanied by increased adrenaline secretion) should logically bring some relief, as was mentioned before.

C. Individual Differences in Psychophysiological Functioning

The concept of arousal or activation has an important place in psychology, and the notion of a continuum of activity intensity has played an explanatory role in fields as diverse as studies of motor performance and theories of personality. The idea probably stemmed initially from Cannon's notions of the role of somatic and endocrine changes in any kind of "emotional" experience, which are stated (Cannon, 1936) to prepare the organism for "fight or flight", and for behaviour requiring the expenditure of energy. Latterly a view prevailed of electrocortical, autonomic, and muscular changes occurring temporally togetther, all of which could be seen as indices of activation. Duffy (1962) and Malmo (1959) are probably the most influential proponents of this point of view, and Malmo has linked arousal with the idea of drive state in learning theory.

However, there has recently been severe criticism of the concept of the unitary nature of arousal. Lacey (1967) reviewed several studies which used pharmacological and surgical methods to demonstrate clear dissociation between somatic and behavioural arousal in both infrahuman and human subjects, and commented that,

> "the evidence seems clear that somatic and behavioural arousal consists of dissociable components mediated by separate neural mechanisms, but that commonly these appear simultaneously". (Lacey, 1967, p.20).

Of more relevance to the work in this thesis is the evidence for dissociation between separate somatic (especially autonomic) components of arousal, and the investigation of individual differences in patterns of physiological responsivity. Because of the diffuse nature of the sympathetic nervous system, there has been a tacit assumption in much work on arousal that its responses in an "emotional" situation would also be diffuse, with all the organs that it innervates responding equally. However, there is evidence that both differences between stimulus situations (especially in the nature of the response involved) and individual differences can combine to produce quite specific patterns of responding.

Arnold (1945) first brought attention to the possibility that different physiological states were associated with different emotions. On the basis of a review of relevant animal studies, she postulated that fear was accompanied by predominantly sympathetic excitation, anger by strong parasympathetic activity, and excitement by moderate parasympathetic activity. In a study designed to study the differential physiological effects of fear and anger, Ax (1953) recorded heart rate, ballistocardiogram, respiratory rate, finger temperature, hand temperature, skin conductance and electromyograph from 43 volunteer subjects. These were recorded in two laboratory situations designed to evoke fear and anger (subjects who did not appear to have experienced these emotions on the basis of behavioural observation and subsequent interview were not included in the study). Seven of the fourteen physiological measures obtained discriminated between anger and fear,

and profile differences between the two states were noted in 42 out of 43 subjects. However, the results did not support Arnold's hypothesis, as diastolic blood pressure increased, skin temperature fell, and face temperature rose more during anger, which is in contradiction to a general parasympathetic reaction occurring in anger. Ax suggests that his results and Arnold's can be better explained on the hypothesis that anger is similar to the sematic reaction to the secretion of adrenalin and noradrenalin, and fear to that of adrenalin alone.

Davis & Buchwald (1957) studied physiological responses of 12 male and 12 female students to a series of slides differing in content. They found that different stimuli quite clearly evoked different patterns of response across all the male subjects, with males responding more than females. Davis (1957) claimed to have found four quite distinct patterns of response associated with mild exercise, auditory stimulation, cutaneous stimulation and response to pictures (though he noted that in this case the content of the picture will affect the response). However, there were large similarities between these patterns and he admitted that they are not distinct entities.

Sternbach (1962) reports a small study with 5 boys and 5 girls who were shown the film 'Bambi'. Skin conductance responses and lacrimation increased notably during the sadder scenes, but generally there was not much consistency in the direction of the responses.

Another important response pattern has been reported by Lacey et al. (1963, 1967), who associate cardiac acceleration with behaviour involving rejection of the organism's external environment, and cardiac deceleration with attention to the environment. This deceleration was associated with a decrease (or relatively low increase) of systolic blood pressure. Increases in other autonomic measures, such as skin conductance were obtained indiscriminantly in both situations. It is possible, however, that the subject's affective state may have been the determinant of this fractionation of autonomic response rather than the psychological demands of the task.

Turning to the studies of individual response stereotypy, most of these have been content to show that this exists, without suggesting causes or trying to relate it to other individual differences. There seems to be a suggestion throughout much of this work (and the work cited above) that these two concepts are somehow mutually exclusive, but there are a few studies showing that this need not necessarily be the case.

Lacey (1950) was first to investigate individual differences in a systematic fashion. He found that normal subjects showed organized patterns of somatic reaction when stressed, which were reliable on retesting on up to 12 occasions and after 300 days. Patterning appeared between different physiological variables as well as between similar ones (e.g. cardiovascular). Lacey & Van Lehn (1952) investigated a group of children aged 6 to 18 with a cold pressor test. The results were fairly repeatable over a period of four years, despite the physiological and psychological changes to be expected in maturing

children. They also noted individual differences in the tendency to show stereotyped response patterns, with some individuals showing the same pattern of response on many occasions, other individuals shifting quite randomly. Hume (1970) has estimated that only a third of normal subjects show specificity in measures of basal level, and only a fifth in basal level and responses together.

Schnore (1959) investigated the consistency of individual differences in physiological reactivity using nine physiological measures. He used two experimental situations and tested subjects in conditions of both high and low arousal, and found stereotyped patterns of responses. Over all subjects, heart rate, blood pressure, respiratory rate consistently differentiated between conditions of high and low arousal.

The reliability of repeated measurements of physiological response has been specifically investigated by Kaelbing et al. (1960). Twelve subjects were given two identical sessions in a conditioning situation with continuous recording of heart rate, respiratory rate, and skin conductance. The test-retest reliability was examined using rank order correlations. Although baseline physiological values were reasonably consistent, responses in general were not predictable.

Vogel (1961) compared the responses (skin conductance, heart rate, respiratory rate, systolic blood pressure, diastolic blood pressure, and finger volume) of groups of children differing in both age and intelligence to two different stressors (loud tones and the cold pressor test). He found evidence for stimulus specificity

and what he claimed was individual response stereotypy; however, this was confined to the conclusion that both age and intelligence affected patterns of autonomic functioning; rather than any demonstration that individuals showed similarity of response to two different stressors. In the younger groups of subjects, intelligence affected autonomic functioning, with significant differences between groups differing in age observed on five physiological measures. He also found that compared with more intelligent subjects, less intelligent subjects made sharper discriminations at the autonomic level between qualitatively different stimuli.

The relationship of stimulus specificity and individual response stereotypy has also been studied by Engel (1960). Using normal subjects, eight autonomic measures, and five different stressors, he was able to show the existence of both types of specificity, though only 8 out of 20 subjects showed statistically significant individual response specificity.

A series of studies by Wenger and his colleagues on the concept of autonomic balance is also of some relevance to the topic of response patterning. Wenger (1942) described how he found factors of autonomic nervous system function and muscular tension in a factor analysis of physiological measures in children. The autonomic factor was found to be normally distributed, and it was possible to assign factor scores to individuals. Deviant scores showed predominance of either parasympathetic nervous system or sympathetic nervous system activity, according to direction, and constituted a situation of

autonomic imbalance. The work with children was replicated on a large sample of young adult males (Wenger, 1948), and regression equations were obtained for estimating autonomic factor scores in adults. Differences were found between the normative group and groups of operational fatigue and neurotic patients (the patients in both groups showing excessive sympathetic nervous system functioning).

Wenger (1966) claimed that the factor estimates were reliable, with high test-retest coefficients over some years. He reviewed data validating the concept covering a wide range of patient groups for whom specific predictions of balance could be made. However, he pointed out that two-thirds of subjects normally showed mixed patterns of sympathetic and parasympathetic activity, and claimed that five consistent patterns of response could be isolated. These were relative sympathetic dominance, relative parasympathetic dominance, autonomic balance, the Beta pattern (a mixed pattern found to dominate in many groups of "psychosomatic" patients), and a different mixed pattern in tubercular patients.

While most of Wenger's work has been carried out on relaxed subjects, Patton (1969) has investigated the effect of stress on autonomic balance in young male adults. They were initially classified as sympathetic or parasympathetic according to their autonomic balance scores, were then exposed to four different stressors, and a non-stress situation, while heart rate, systolic blood pressure, skin conductance

and skin temperature were recorded. The changes in overall sympathetic nervous system activity during stress (regardless of the autonomic measure involved) were relatively consistent. Differences were also found between the sympathetic and parasympathetic groups, and these were attributed to constitutional differences in autonomic activity and to the greater reactivity of 'sympathetic' subjects during stress.

This approach to the study of physiological responses can be seen as complementary to the studies previously described. The factorial methods used emphasise such communality as there is between different physiological measures, and the use of a large group of "normal" subjects to provide normative data is in marked contrast to other work in this field.

Graham and his co-workers (Grace & Graham, 1952) put forward a hypothesis relating psychosomatic disorder, physiological response specificity and attitudes. This hypothesis stated that there is a specific relation between the attitude (i.e. what a person feels is happening and what he intends doing about it) that a patient develops towards a disturbing life state and the diseases or symptoms that he develops. They assume that a different physiological process underlies each psychosomatic disease, and that each particular attitude is associated with its own set of physiological changes. A series of experiments testing various aspects of this hypothesis has been carried out. Graham, Stern & Winokur (1958) induced attitudes in hypnotised normal subjects that they claimed were associated with urticaria and Raynaud's disease -- the two attitudes being that the person sees himself as mistreated without wishing to take retaliatory action on

the one hand, and wishing to take some hostile action on the other. They argued that since warming of the skin is part of the symptomatology of urticaria and cooling is typical of Raynaud's disease, the induction of these two attitudes should show changes in skin temperature in the appropriate direction. This prediction was substantially confirmed.

In a further study, Graham, et al. (1962) compared the physiological responses of hypnotised normal subjects to the suggestion of the attitudes associated with both urticaria and hypertension (the attitude suggested for hypertension being one of being continually on guard for danger). Skin temperature, systolic blood pressure, diastolic blood pressure, pulse and respiratory rates were monitored.

It was predicted that skin temperature would rise more with the urticaria suggestion than with that for hypertension, and that diastolic blood pressure would rise more with the hypertension than with the urticaria suggestion. These predictions were confirmed, and furthermore no differential effects of the two attitude suggestions were found in systolic blood pressure, heart rate, or respiratory rate.

Another study in this series (Stern et al. 1961) replicated the 1958 study and also included the response to the hypertension attitude. In addition, an attempt was made to introduce the urticaria attitude (with its consequent rise in temperature) when the subject's skin temperature was falling, and conversely, the Raynaud's disease attitude was induced whenever skin temperature was rising, thus providing a more stringent test of the hypothesis. They found that the diastolic blood

pressure rise with the hypertension attitude was more than the rise with the two other attitudes. When significant differences between the slopes of the temperature curves during the control period had been taken into account, the changes during the attitude induction period followed the prediction -- however, they were not successful in producing an absolute rise consistent with the urticaria attitude following a fall in the control period.

D. The Relationship of Physiological Measures to Measures of Personality

1. Theoretical Contributions

The first attempt at a causal explanation of human personality differences in terms of constitutional factors was probably due to the Roman physician Galen, whose theory of the humours divided people into the Sanguine, Melancholic, Choleric and Phlegmatic. These temperaments were said to be caused by the strength of the blood, the overfunctioning of the black bile, the predominance of the yellow bile and the influence of the phlegm respectively. This idea persisted in the West throughout medieval times and the Renaissance, to find a new advocate in Kant in the 18th century. Wundt (1903) saw these temperaments as the interaction of two dimensions, one being the strength of the feelings, the other their speed of change. Moreover, these two concepts of strength and speed of change have persisted in one form or another in the two personality theories described briefly below.

a. Pavlov's Theory of Personality

In his work on conditioning, Pavlov had always been aware of the marked individual differences between dogs. This was ascribed to constitutional differences in central nervous system activity, and late in his life Pavlov became interested in the application of these ideas to humans, especially psychiatric patients. Gray (1964) has provided a detailed review of this and later Russian work, and has attempted to relate some of their concepts and the results of experimental studies to work carried out in the West.

Pavlov postulated that central nervous system activity, and therefore behaviour, could vary along the three independent dimensions of strength, mobility and equilibrium of the two opposing processes of inhibition and excitation. Teplov (Gray, p.274) distinguished between strong and weak nervous systems (referring only to the excitatory process), and has shown that the strong nervous system acts to damp down stimulation, while the weak nervous system acts as if it amplified it. This manifests itself in such measures as sensory thresholds (lower in the weak nervous system), reaction time (faster in the weak nervous system) and responsiveness to the administration of caffeine, which alters sensory thresholds in the weak nervous system but not in the strong.

b. Eysenck's Theory of Personality

It will be apparent that a limitation of Pavlov's theory is that individual differences in central nervous system activity are related only to limited "molecular" acts of behaviour, rather than to the broader personality traits which are customarily used to describe people and with which Western psychology has mainly concerned itself. In his theory of personality, Eysenck (1960) has tried to integrate the various levels of description of personality on a hierarchy, borrowing on the ideas of Pavlov mentioned above, and on Hullian learning theory. Eysenck's theory, with its two orthogonal dimensions of Neuroticism and Introversion-Extraversion, has been so well documented (Eysenck and Eysenck, 1969; Eysenck, 1955) that it is not proposed to review it or the many experimental studies cited in support of the theory. However, Eysenck (1967) has attempted to relate individual differences in neuroticism and emotionality to sympathetic nervous system activity and the concept of arousal, and it is this aspect of his theory which is most relevant to the work described in the following chapters.

Eysenck has argued that differences in both extraversion (1963) and neuroticism (1967) can be linked to variations in physiological arousal. However, Claridge (1967) put forward the view that each of these dimensions could be linked with separate dimensions of arousal. He postulated the existence of two functionally related arousal mechanisms, a tonic arousal system and an arousal modulating system.

On an operational level, changes of the functioning in these systems were related to changes in the sedation threshold and the spiral after-effect, respectively. In normal subjects and in neurotics, these two mechanisms are closely related, with cyclothymics showing high arousal modulation and low tonic arousal, and dysthymics the opposite. Claridge postulated that dissociation between the two mechanisms underlies the psychoses, with weak arousal modulation and high arousal being characteristic of the paranoid patient, and low arousal and high modulation characteristic of withdrawn, non-paranoid psychotics. A personality dimension of "anxiety drive" is identified with tonic arousal, and introversion-extraversion with the arousal modulating system. Claridge emphasised that at least two trait clusters, "behavioural" and "social" extraversion may make up the introversion-extraversion dimension. However, it should be said that the research on which Claridge based his ideas was mainly carried out using psychiatric patients as subjects, and the extension of his theory to account for personality differences in normals is an indirect one.

2. Experimental Work

Although the investigation of individual psychophysiological differences has shown that they are persistent and in some way characterise the individual, the study of their relationship to personality measures has on the whole been disappointing in the inconsistency of the results obtained. Several reasons for this are apparent. The first is the complete lack of standardisation

of psychophysiological measurement techniques from one group of workers to another. Allied to this is the selection of inappropriate techniques; for instance, the wide variation of the electrical properties of skin resistance electrodes (Lykken, 1959) may introduce errors in measurements and thus render comparisons between studies impossible. The second reason is the often arbitrary choice of personality measures and a seeming lack of appreciation of their appropriateness to this kind of study. The third criticism is the inadequacy of much of the reported research in terms of numbers of subjects, appropriate controls and statistical treatment of results.

Duffy (1962) reviewed work up to that date on behavioural correlates of individual differences in arousal, including biochemical, electrodermal, cardiovascular, EEG, and electromyographic studies. She concluded that "the studies ... give support to the conclusion that differences between individuals in activation are basically differences in responsiveness or excitability"; however this simple relationship has not been accepted by later workers.

Not all of the studies have used questionnaire measures of personality. Brown (1925), in the earliest study known, found no connexion between skin conductance responses to stimuli in children, and estimates of 'character' by their teachers. Similarly, Darling (1940) obtained ratings of behaviour characteristics in children with measures of skin conductance and blood pressure, and factor analysed the data. He obtained two factors which he denoted as parasympathetic and sympathetic, judging by their loadings on the autonomic measures.

None of the behaviour ratings showed large loadings on these factors; however, the inter-rater reliability was exceedingly low.

Darrow and Heath's study (1932) was the first to use a battery of psychometric tests. Three groups of University of Chicago volunteers were given the Thurstone Neurotic Inventory and a test of introversion-extraversion. Blood pressure, respiration, skin conductance and arm movement were measured under conditions of warning of electric shock, the count-down to it, and the shock itself. Most of the correlations obtained were low, but skin conductance and blood pressure responses to shock, and the adaptation of skin conductance responses were related to 'excitability'. From both the physiological and psychological data a "hyporeactivity-hyperreactivity" factor was tentatively identified.

Wenger (1948) obtained completed Guildford Personality Inventories from over 300 aircrew, who had also completed a psychophysiological test battery from which measures of autonomic balance were obtained. Forty-six out of 325 possible correlations between individual physiological and psychological variables were significant at the 5 per cent level or beyond, which is more than would be expected by chance. All but four of the personality factors were correlated with two or more of the physiological variables. The autonomic factor score correlated with personality factors N (lack of nervous tension), D (depression), C. (emotional instability), O (Objectivity) and CO (tolerance of others; cooperation). Two other physiological factors (named blood-sugar level and thyroid activity bore no significant relation to any of the personality variables.

In contrast, Terry (1953) found only four significant correlations out of a possible 87 between psychophysiological factors and psychological measures, using the Guildford-Zimmerman Temperament Survey. In an unpublished thesis, Berger (1958) investigated the relationship between a battery of personality tests (Cattell 16 Personality Factor Questionnaire, the Edwards Personal Preference Schedule and the Blackie Pictures Test), and basal and response measures of skin conductance, heart rate and respiration rate under three stresses. His subjects were a group of 30 male psychiatric patients and a group of 27 college students. The correlations between 40 psychophysiological and psychological variables were factor analysed using the centroid method, and rotated to simple structure. Five factors were felt to be sufficiently similar in the two samples to appear to be cross-validated. Several inter-relationships between physiological reactivity and personality were found. The skin conductance response was linked primarily to the inhibition of expression of impulses. An increase in respiration rate under stress was found to be related to anxiety and hostility, whereas passive dependence was associated with decreased respiration rate. Dependence was linked to a decrease in heart rate under stress.

Dykman et al. (1959) studied physiological reactions to novel stimuli and the relationships between physiological reactions and psychological variables in a group of 40 medical students. They found that subjects with high TMAS scores tended to show higher overall autonomic reactivity, but the differences between high and low anxiety

groups was not significant. They also found that high scoring subjects on the K scale of the MMPI (a scale said to reflect the type of defensiveness whereby a person denies abnormality in himself and in others) showed higher autonomic reactivity. In a further study (Wilson and Dykman, 1960), spontaneous skin conductance activity in medical students was also found to correlate with the K scale and the TMAS.

There have been several attempts to verify Eysenck's prediction that questionnaire scores on neuroticism would be correlated with 'instability' of the autonomic nervous system. Bronzaft et al. (1960) found no relationship between skin conductance response in an avoidance situation and the 'N' scale of the Maudsley Personality Inventory in a sample of 46 college students. In a more extensive study, Fahrenberg and Delius (1963) gave a sample of 94 male psychiatric patients the MPI and the Rorschach and recorded a variety of autonomic measures during relaxation and under stress. A factor analysis of the data revealed three identifiable factors. One of these loaded almost entirely on cardiovascular variables, one loaded on introversion, neuroticism, heart rate, blood pressure, response to stress and breath-holding time (identified as anxiety and its associated autonomic components), and a third mixed factor which seemed to reflect good homeostatic regulation.

Burdick (1966) failed to find any association between the Neuroticism Scale of the MPI and spontaneous skin conductance activity. He recorded skin resistance during relaxation in 27 male college students.

No difference in N scores was found when the group was divided into "stable" and "labile" subgroups on the basis of their electrodermal activity, nor was there any correlation between the psychological test scores and spontaneous fluctuations in skin conductance (SCSF). However, a very high current (70μA) was used to measure skin conductance, and this must have caused tissue damage (Edelberg et al. 1960), which would have introduced error in the skin conductance measures. Also, the relatively insensitive method of counting the number of 30 second periods containing a SCSF was used, which would be insensitive to frequent spontaneous activity (note that Lader and Wing, 1966, report a mean SCSF rate of 2 per minute in normal relaxed subjects).

Kelly and Martin (1969) found negative correlations between the physiological response to a stress of mental arithmetic and neuroticism scores in a group of chronically anxious, neurotic and normal subjects. A factor analysis of their data showed a definite anxiety factor, loading on neuroticism, introversion, basal heart rate, blood pressure and forearm blood flow.

Another personality theorist who has studied the relationship between physiological measures and personality traits is Cattell. In contrast to Eysenck, however, physiological measures do not have any explanatory role in his theory of personality, but he has reported on several occasions that factorial studies of psychophysiological measures yield factors which seem to relate to personality source traits derived from psychological measurements. Thus it is appropriate

to consider his work here rather than in the preceding review of theories.

Cattell (1955) claimed to have found seven general personality factors with somatic associations. Most of the studies he cited have been carried out using "P-technique" analyses, i.e. the examination of covariation of certain measures in individuals on repeated testing. It has been claimed that the correspondence between factors derived from this data and those derived from "R-technique" (the study of individual differences in a group of individuals examined on one occasion) may not be exact, since the variation in individuals may be so small that patterns of change will not be detectable. However, for the analysis of psychophysiological measures, which are known to be labile and to show marked individual differences, such criticisms of "P-technique" do not seem to be relevant. Of these seven personality factors, five of them seem to be related to recognizable personality factors obtained by R-technique, which appear in the 16 PF Personality Questionnaire (described in chapter 5). A brief summary of the findings is presented in table 2:1.

Table 2:1

P.U.I.* no	Description of Factor	Related Personality Factor	Physiological Variables
1	Parasympathetic resilience (good adjustment to stress).	H	Large skin conductance deflections; Low skin conductance; Low pulse rate; High urine volume.
2	Desurgency vs. surgency (low cortical alertness).	F (-)	Alkalinity of saliva; Low body temperature; Low cholinesterase in serum.
4	General Adaptation Syndrome (Stage 1) (Stress response).	C	Small skin conductance deflections; High 17-ketosteroid; High cholinesterase.
5	Adrenergic, cyclothymic state.	A	High blood sugar; large skin conductance downward drift in relaxation; High pulse rate.
7	Basophil/Neutrophil	O?	Few eosinophil cells; Few basophil cells.

* P-Technique Universal Index.

Cattell et al. (1955) found that factor H is largely genetically determined, and Cattell hypothesized that in this case the personality trait is largely determined by the physiological "resilience", which is constitutional, rather than vice versa.

In the same paper, Cattell also suggests that a factor named Autonomic Inactivity (high serum cholinesterase, low pulse pressure and saliva, low skin conductance) is associated with personality factor L (Trusting-suspicious), but does not regard this as well-confirmed.

Cattell (1966, p.683) claims that most factor-analystic studies of psychophysiological variables have not succeeded in distinguishing between trait and state patterns, and notes that the "P-technique" of studying longitudinal changes will theoretically yield factors representing dimensions of state. Cattell (1966, p.362) has discussed this distinction between trait and state factors. In the case of psychophysiological measures, however, it seems reasonable to assume that state and trait patterns will be similar, since it is difficult to conceive of such wide anatomical (or structural) differences between normal human beings that would cause really marked differences in the patterns of change found in physiological measures. It seems convenient at this time, therefore, to regard the distinction between psychophysiological trait and state as mainly one of time scale.

Other investigators have looked at the correlations of yet other personality traits and physiological measures. Two of the favourites are the "ego strength" scale of the MMPI and indices of "emotionality". Greenfield et al. (1963) compared low, middle and high ego strength groups in their physiological responses to tones of various loudness; latency of response, time to peak magnitude and per cent recovery time measures were used. There was no systematic trend in their results, except that possibly the high ego strength group showed generally "slower" responses with longer times to peak response and slower recovery time in some of the measures. However, this study can be criticised as the experimentally obtained group of subjects contained both psychiatric patients and normal subjects, and it is possible that differences in anxiety between the groups could have affected the results. In a subsequent study employing only normal subjects, Roessler et al. (1965) found that high ego strength subjects showed greater response amplitude to sound and light stimulation, but did not discuss why this personality dimension should be associated with differences in psychophysiological response. A more recent study by this group (Roessler and Collins, 1970) compared physiological responses to stressful and bland films in groups of students differing in ego strength. The high ego strength group responded more in skin conductance and heart rate to both stressor and bland films, and showed a differential response to the stress film compared with the bland one.

There are several reports which suggest that a tendency not to express emotion is associated with greater psychophysiological responsivity. Jones (1950) noticed that "children who are the most overtly excitable are frequently the least reactive on the galvanometer" and reported that adolescents showing low skin conductance responsiveness in the stressful situation of a free association test were rated as showing more attention-seeking, talkative, animated and assertive behaviour. The study by Berger (op. cit.) suggested that increased electrodermal responsivity was linked to the inhibition of expression of impulses. Learmonth et al. (1959) tried to relate fluctuations in skin potential during stress with various personality scales from the MMPI in a group of 20 nurses. They found correlations between skin potential fluctuations and the MMPI Hysteria, Psychopathic Deviancy, and Hypochondriasis scales, and suggested that increasing tendencies to express feeling are associated with less physiological reactivity.

Schachter and Latané (1964) found that sociopathic individuals manifested higher heart rates during several experiments than more emotional subjects, and also that sociopaths were more autonomically responsive to stress, and more sensitive to adrenaline. Valins (1967) classified a group of male students into unemotional and emotional groups and compared heart rate responses while anticipating and receiving both neutral (tone) and unpleasant (shock) stimuli. He found that unemotional subjects increased heart rate while anticipating shock, while the heart rate of the emotional group decreased. However, no

differences were found between the groups in their reaction to either type of stimulus, or in the anticipation of neutral stimuli. Thus it appears that only in the emotional situation of anticipating shock (and, maybe, emotional situations in general) are the differences between emotional and non-emotional people made manifest. Lazarus and Alfert (1964) reported that people with a tendency to deny threat (as measured by the MMPI) denied affective disturbance when watching a stressful film but nevertheless showed increases in heart rate and skin conductance.

Alfert (1967) has advocated the use of idiographic approaches in investigating the effect of personality differences on response to threat. Essentially, this is an approach which seeks to discover relationships within the individual, i.e., does he react to one situation more than another? She argues that nomothetic methods (i.e., the comparison of groups of subjects differing in a particular personality measure, or the division of a group of subjects into high and low reactors) will obscure the relationship between personality factors and responsiveness to a particular stimulus. This is due to individual differences in autonomic responsivity, and also because an individual's characteristic way of physiologically responding to situations generally will account for much of the variability in such an experimental design. To demonstrate this, an idiographic analysis showed that subjects responding more to direct threat (shock) than to a vicariously experienced threat (watching a film of industrial accidents) were more extrovert, self-confident, dominant and at ease in

social relationships, while the opposite group were introverted, anxious and inhibited in impulse expression.

Opton and Lazarus (1967) developed the reasons behind an idiographic approach in greater depth. They enumerated several possible sources of variation that could contribute to the physiological response to psychological stress. Personality traits would be one factor influencing the cognitive appraisal of the situation, and differences in personality could lead to differing appraisals of the situation as more or less threatening, and hence to differing degrees of physiological response. At the same time, however, these personality differences could hypothetically be related independently to constitutional physiological differences which would exert an independent influence on the physiological response; in addition, these constitutional differences could be quite unrelated to any psychological measures and still influence the final response. If the main question asked in a study is - does a particular personality trait imply a particular sensitivity to some types of stress rather than to others, then the most promising solution is the intra-individual design where an individual's responses are compared in several situations. By analysing selected data using both idiographic and nomothetic methods the authors demonstrated the efficiency of the former technique in answering this question.

E. Summary and Conclusions

It is difficult to draw many firm conclusions from the studies on asthmatics, since such a wide range of techniques were used for personality and psychophysiological assessment, and some of them suffered from deficiencies in sampling or did not include control groups. However, there does seem to be a consensus that
a) emotion is an important aetiological factor for many asthmatics;
b) while there is no specific personality type associated with asthma, asthmatics often possess traits (such as neuroticism and hostility) which could cause them to behave 'emotionally' in many situations where others would not. This could have two effects on their asthma. One is to enhance subjective distress during an attack of asthma; the other is to create physiological changes (in the Autonomic Nervous System) which would interact at a somatic level with the physiological changes associated with an attack of asthma.

Although it has been fairly convincingly demonstrated that emotional events can influence respiratory function, in both asthmatics and non-asthmatics the conclusions to be drawn on the role of the autonomic nervous system in asthma are less clear. Although the paper by Robinson showed that differences in autonomic reactivity could play an important part in the susceptibility to attacks of asthma, it cannot be said that the studies by Wenger and Hahn have demonstrated more than the fact that there may be homeostatic abnormalities in the autonomic nervous system in asthmatics. Until more normative data is available, for adults as well as children, these psychophysiological findings cannot be interpreted as more than suggestive.

The"attitude specificity" studies of Graham et al. are the most encouraging empirical work in the psychosomatic field, since their concept of a predominant attitude towards life events seems to be equivalent to the idea of a personality trait, and their physiological findings in both normal and "psychosomatic" subjects are remarkably consistent. It is unfortunate that they have not extended their empirical studies to a consideration of asthma.

Is there any other consistent evidence for an association between personality traits and psychophysiological measures? Two main points seem to emerge from the work reviewed. One is the relative lack of success in verifying Eysenck's prediction that neuroticism is associated with heightened autonomic activity. While the studies using patient group (Fahrenberg and Delius, Kelly and Martin, op. cit.) have found an association between high neuroticism questionnaire scores and heart rate, the studies using normal subjects and electrodermal measures (Bronzaft et al., Burdick, op. cit.) found no such associations.

The other notable point is the suggestion that lack of emotional behaviour is associated with heightened physiological activity. Confirmation of this idea seems to emerge from all the studies that have found a personality dimension of "defensiveness" to be associated with high autonomic activity (Berger, Dykman et al., Wilson & Dykman, Schachter and Latane, op. cit.). A partial explanation for this could be given in terms of Lacey's hypothesis that environmental rejection is associated with increased heart rate, since the idea of "defensive"

behaviour in the experimental situations used in these studies seems necessarily to entail a rejection of external events. But this would not explain the results where electrodermal variables have shown increases in activity. It is tempting to interpret this phenomenon in Freudian terms; could the inhibition of unacceptable feelings lead to increased physiological activity and perhaps later lead to symptom-formation? These studies will be interpreted as possibly somewhat unexpected, but none the less empirical evidence for this; and the importance of this conclusion in eliciting the role of psychological factors in asthma is great indeed.

CHAPTER 3

OVERALL DESIGN OF THE STUDY

A. Initial Considerations

The aim of the research reported in this thesis was to investigate physiological reactivity and personality traits in asthmatics. Since it seemed likely that any differences which might emerge between asthmatic and non-asthmatic subjects would be on traits related to anxiety and neuroticism it was decided to include two groups of normal (non-anxious) and neurotic (anxious) subjects as control groups bi-polar to the dimension under study. For reasons of time, it was not possible to match each asthmatic subject with both a normal and neurotic control, so an initial design was adopted whereby each asthmatic subject was matched with either a normal or neurotic control subject, with the group of the control subject chosen on a random basis. The control subject was to match the asthmatic he or she was yoked with on the basis of age, sex, and social class. One mismatching of sex occurred, when a female asthmatic was matched with a male neurotic of the same age and social class, and a male asthmatic was yoked to a female normal subject.

This initial design was later expanded to include a further 24 asthmatics who were not matched with controls; in addition another group of asthmatics was formed, comprising 14 individuals who had been referred to the Royal Edinburgh Hospital for a psychiatric consultation in connection with their asthma. There are methodological reasons,

which will be discussed later, for these subjects to be considered as a separate experimental group. The criteria laid down for inclusion in each group, and the methods by which individuals were selected for participation on the study are described below. Table 3.1 shows the number of subjects in each group, divided by sex, and their mean ages.

TABLE 3.1

"Random" Asthmatics

Group	Source of sample	mean age	male	female
1	Northern General Hospital Acute Episodic Sample I	39	4	8
2	Northern General Hospital Chronic Sample I	42	4	8
3	City Hospital and Royal Infirmary	37	5	5
4	General Practitioners (not referred to hospital)	32	3	7
5	Northern General Hospital Acute Episodic Sample II	36	5	7
6	Northern General Hospital Chronic Sample II	39	2	10
"Referred" Asthmatics				
7	Referred to Royal Edinburgh Hospital	37	8	6
Control Groups				
8	Neurotic Controls	38	9	13
9	Normal Controls	37	7	15

	Men	Women
68 "random" asthmatics	23	45
14 "referred" asthmatics	8	6
22 "neurotic" controls	9	13
22 "normal" controls	7	15

B. Description of the Experimental Group

1. The "random" asthmatic group

One of the main problems confronting anyone who wishes to select a group of probands suffering from a particular disease is to ensure that they are in fact representative of the population in question. Regrettably, as noted in the review of literature, the criteria for the selection of asthmatics in many previous studies have often not been made explicit, and when they are, it has too often been the case that the subjects chosen are a far from random sample, thus seriously limiting the validity of the findings.

Recognising these difficulties, special efforts were made to ensure that our subjects were a representative sample of the population of "asthmatics in Edinburgh". Ideally, we should have gone from door to market research door, hunting out sufferers, but this was scarcely feasible. So instead, several potential sources were used. These were

a) The register of asthmatics attending the respiratory diseases clinic at the Northern General Hospital. This register divides asthmatics into two groups - the "chronic" group who suffer from asthma almost continuously, most of whom are prescribed prednisolone (a corticosteroid drug) to alleviate their distress, and the "acute episodic" group whose pattern of respiratory distress is episodic in nature. Relatively few of these are prescribed steroids.

b) The register of asthmatics at the City Hospital.

c) The register of asthmatics at the Royal Infirmary.

d) Asthmatics not attending a hospital clinic but known to their general practitioners.

Clearly, this is not a random sample. We do not know the proportion of subjects obtained from each source in this investigation is the same as the proportions in the total population of "asthmatics in Edinburgh". Nor can it be said that there are no other sources of asthmatics remaining unsampled - such as the (probably hypothetical) group of those not known to any medical practitioner. But it can fairly be said that these are a representative sample of asthmatics, and certainly not selected *a priori* on grounds of psychiatric morbidity. We have also avoided studying solely individuals who are *currently attending* a clinic for their asthma, by sampling from the registers at these clinics. This is particularly important in the light of the observation by Zealley (1971, *op.cit*) that "clinic attenders" score higher on neuroticism scales than a group of 'non-clinic attending' asthmatics.

2. *The "referred" asthmatic group*

During the two years while the data was being collected, the psychiatrists involved inevitably saw some asthmatics who had been referred to them on the grounds that a psychiatric opinion might be helpful in the management of their asthma. It was thought that this group might be equivalent to those used in studies which had confined themselves to asthmatics known to psychiatrists, or undergoing treatment with them.

3. Neurotic control subjects

Half of the asthmatics in groups 1 - 4 (chosen on a random basis) were matched with neurotic controls, of similar age (±8 years), sex and social class. The criteria for inclusion were that

a) Each subject should have been an in-patient in the Royal Edinburgh Hospital during the year preceding participation in the study, and

b) They were assigned a principal diagnosis of neurosis with anxiety as a prominent feature.

c) They were currently attending the Royal Edinburgh Hospital as out-patients.

d) Mood disturbance should have been noted as a principal symptom.

Names of suitable subjects matching these criteria were obtained from the medical records at the Royal Edinburgh Hospital.

4. Normal control subjects

These were selected by asking friends of the researchers (who were not themselves working in the psychological or medical fields) to nominate healthy acquaintances of theirs of the required age, sex, and social class. These were matched with the remaining asthmatics in groups 1 - 4.

C. Initial Contact with the Subjects

The normal group of subjects were contacted directly by the experimenter to confirm that they would be willing to take part in the research. The other subjects were contacted after their general practitioners and consultants had been notified that these patients were being asked to take part in the research. No objections were received from any medical practitioners. Few of the subjects refused to take part in the study;

when they did, it was usually because they had moved away from the centre of the city and it would be difficult for them to attend. When this occurred, another name was drawn at random from the same source as the non-participating individual and was contacted in the same way.

D. Procedure

The procedure followed on each of the two visits is shown in Table 3.2. The asthmatics in groups 5 and 6 were seen on only one occasion, and instead of the interview filled in a medical questionnaire which was designed to elicit the same information that was obtained from the other subjects during the interview. These interviews were conducted by the two psychiatrists concerned in the study, and its purpose was to obtain information about the subject's health, both physical and mental, and to rate the presence of certain personality traits. The results of this interview and questionnaire data are not reported in this thesis (see Aitken et al. 1970); however, such items as the subjects' ages were extracted from the interview protocols.

TABLE 3.2

Timetable of assessment procedure

Session 1	Session 2
*Interview (up to 60')	*Interview (up to 60')
Introduction to laboratory (5')	Forearm blood flow (25')
PGR Habituation (35')	Breathing against respiratory stress (25')
Breathing against respiratory stress (25')	
*Assessment of subjective distress (10')	*Assessment of subjective distress (10')
Psychological Testing (30') (Eysenck Personality Inventory, Hostility and Direction of Hostility Questionnaire, Taylor Manifest Anxiety Scale)	Psychological Testing (45') (16 PF Questionnaire)

Items marked with an asterisk are not reported in this thesis.

Subjects seen only on one occasion were given the tests in the following order

*Questionnaire (up to 45')

Introduction to Laboratory (5')

PGR Habituation (35')

Breathing against respiratory distress (25')

*Assessment of subjective distress (10')

Forearm Blood Flow (25')

Psychological Testing (up to 75')

This timetable was adhered to as rigidly as possible. Changes occasionally had to be made, and these could necessitate the subject attending on a third occasion. Wherever possible, though, the psychophysiological tests were carried out on the first two occasions in the order stated. It is appreciated that no attempt was made to control for the possibility of adaptation to the laboratory situation. Sabshin et al.(1957) have shown that exposure to a laboratory situation by itself can be considered as stressful. However, deliberate attempts were made to put subjects at their ease in the unfamiliar surroundings, and the harmless nature of the psychophysiological tests was made quite explicit. A measure of the success in allaying anxiety was that no subject dropped out of the study after the first laboratory session.

The psychophysiological testing took place in a small cubicle (10' x 6') attached to the Psychophysiology Laboratory in the University Department of Psychiatry. It was air-conditioned and maintained at a constant temperature of 22°c. The humidity was monitored but not controlled in any way. The cubicle was in constant two-way communication with the experimenters in the laboratory through a high quality intercom system, and it was made quite clear to the subjects that they could communicate with the experimenters at any time. The subjects were seated in a chair for all the tests except the forearm blood flow, when they lay down on a couch in the cubicle.

Before the first psychophysiological test (normally the PGR habituation test) and before any electrodes were attached, the subjects were seated in the cubicle and the following introduction was played to them from a pre-recorded tape.

"The test we are going to do with you is absolutely straightforward, so far as you are concerned, and it will cause you no difficulty or trouble at all. You will simply be asked to sit in a comfortable chair, and a number of wires will be lightly fastened to you. You will feel nothing through these wires -- no shocks or anything else.

In the second part of the test, you will also be asked to breathe through a special face-mask, so that we can measure your breathing. You will of course be breathing only ordinary air all the time.

The test is in <u>two</u> main parts, each lasting about 20 minutes or so. During the first part, you will simply be sitting in a quite little room and you should just relax and take it easy. After the first few minutes, you will hear a fairly loud sound lasting one second. Don't worry about this -- you don't have to do anything at all about it. This sound will be repeated at intervals during this first half of the test.

During the <u>second</u> half of the test, you'll be fitted up with the face-mask I mentioned, so that your breathing can be measured. The measurements we make have to be accurate, so the face-mask is quite a firm fit on your face -- but you should not find it uncomfortable. At certain points during the breathing test, you may notice that you have to breathe with a little extra effort for a minute or two. This is all part of the test, and the whole procedure is under careful control, so don't worry.

At the very end of this second part of the test, we ask you to tell us just how hard you found the different bits of the breathing test to do. To do this, you will be given a piece of paper with a line drawn on it. You are asked to imagine that, if you made a mark on the line at

the extreme left hand end, that would mean that you felt absolutely no difficulty with your breathing at all. A mark at the extreme right end of the line would mean that breathing was completely impossible, it was so difficult. Now of course, we won't be making the breathing as hard as that; but the ends of the line have to represent the extremes of breathing -- absolutely no trouble at all at the left hand end of the line, completely impossible to breathe at the right hand end. And of course a mark somewhere in the middle would mean breathing of moderate difficulty. You are free to mark the line wherever you wish, so long as you bear in mind that we are really asking you to make a comparison between the different degrees of breathing difficulty that we expose you to.

So that we don't affect the measurements in any way, we avoid coming into the room where you'll be sitting. But we are in complete contact with you all the time by means of an intercom system -- if you say anything to us, we can hear you; and we can talk to you without having to come into the room. Naturally, once the test has started, we hope you'll not need to say anything, so that we can complete the test as quickly as possible.

Finally, we will be asking you to fill in one or two questionnaires that will help us to make the best use of the results of the laboratory tests.

Well, that is all I need to say. If you have any questions to ask at all, please do not hesitate to do so. If you'd like me to explain anything again, I shall be very happy to do so. As I said before, the whole test is simple as far as you are concerned, and should not upset you at all".

The subject was then prepared for the psychophysiological tests. The procedure followed for each of the tests is reported in chapters 4 (Habituation of the PGR and forearm blood flow measurement) and 6 (response to breathing against a respiratory stress).

CHAPTER 4

TWO PSYCHOPHYSIOLOGICAL TECHNIQUES FOR THE ASSESSMENT OF ANXIETY: HABITUATION OF THE PSYCHOGALVANIC REFLEX AND FOREARM BLOOD FLOW

A. Introduction

The two assessment techniques in this chapter are grouped together because they both represent an important advance in psychophysiology. They have in common that they both attempt quantitative measurements of physiological indicants of anxiety, and have met with some success in this field. Thus they seemed well suited as tools to help with the enquiry into the relationship between physiological and psychological measures. Initially, the establishment of differences between the two control groups would be an important and necessary demonstration of their validity as measures of physiological indicants of anxiety. The results of the asthmatic group and the relationship between these physiological measures and psychometric indicants of anxiety should allow a fuller assessment of the role of anxiety in the personality of asthmatics.

B. Habituation of the Psychogalvanic Reflex (PGR)

The technique used to assess habituation of the psychogalvanic reflex (PGR) has been discussed in detail by Lader and Wing (1966), who also reviewed the relevant psychiatric, psychological, and neurophysiological literature extensively. Thus, only their findings and subsequent studies will be discussed here. They recorded four physiological measures simultaneously, skin conductance, (SC), finger pulse

volume, pulse rate, and forearm extensor electromyograph (EMG), for a period of 32 minutes. After 10 minutes rest the stimulation procedure began. Twenty identical auditory stimuli consisting of a 1000 Hz tone at 100 db were played to the subject, at random intervals of between 45 and 80 seconds.

Their first study was carried out on 64 university students, given doses of cyclobarbitone or placebo in a balanced design. They found that when the PGR was converted to log conductance units, succeeding responses decreased linearly with the logarithm of the stimulus number. Linear regression analysis was therefore used as a method of quantifying the rate of habituation. It is possible to test the significance of each individual's regression coefficient, using analysis of covariance, and it was found that about two-thirds of the student subjects tested were 'habituators'. Cyclobarbitone produced significant changes in the amount of spontaneous activity, in the change in SC level during the experiment, and in the rate of habituation. There were fewer systematic changes in the cardiovascular and EMG variables, except that a paradoxically higher pulse rate was observed with cyclobarbitone when compared with the placebo group.

A similar study was carried out on 20 patients with anxiety states and with 20 normal controls matched for age and sex. The conductance variables differentiated between the groups significantly, the patients demonstrating a greater number of SC fluctuations, higher SC level, and a slower rate of habituation. All the normal subjects, but only six of the patients, were habituators. Pulse rate also differentiated the

groups significantly. A 'composite physiological activity' score was derived from a discriminant function analysis of the data, and this was found to correlate with ratings of overt anxiety made just before the physiological recordings.

Lader and Wing discussed their results in terms of the concept of arousal. They postulated a mechanism for the production of morbid anxiety (which they assumed to be the experience of over-activity in the central nervous system), based on their finding that habituation seems to vary with the level of arousal, being less with high physiological activity. A novel stimulus causes a rise in arousal, this rise being larger with low levels of arousal. In relatively non-aroused subjects, habituation would be quite fast, but in already highly-aroused subjects there will be little habituation and repetitive stimulation would increase arousal even more, thus further decreasing the possibility of habituation and triggering a "positive feedback" system. In highly anxious individuals this could lead to chronic anxiety and to panic attacks, especially with symbolic real life stimulation of a highly anxiety-evoking nature.

Since the publication of lader and Wing's monograph, several studies report further investigations of the variables involved in PGR habituation. Lader (1967) studied a mixed group of 90 patients with diagnoses of anxiety-with-depression, anxiety state, agoraphobia, and monosymptomatic phobias, in an attempt to relate these syndromes to his previous work. In general, the specific phobics were rated as less anxious and responded physiologically by habituating in a similar

manner to normal subjects. In all subjects, habituation rate correlated with general anxiety, and the magnitude of the first PGR response correlated with situational anxiety level. Overt anxiety (rated by the experimenter immediately before the test) correlated with most physiological variables. Hagdahl et al. (1967) showed that PGR and heart rate response habituated to both a simple stimulus (800 Hz tone at 70 db) and the more complex stimulus of a perceptual conflict test based on the Stroop Colour Test (Stroop, 1935). Israel (1966) showed that when a group of subjects were divided on a cognitive test into 'levellers' (those who tended to see slightly differing visual stimuli as equivalent) and 'sharpeners' (who perceived differences more readily), the 'levellers' showed lower PGR reactivity to a repeated visual stimulus, and habituated quicker than the 'sharpeners'.

Koepke and Pribram (1966) confirmed that the speed of PGR habituation was inversely related to spontaneous SC activity in a group of college students, and showed that the duration of the stimulus (a 2 or 20 second tone) did not affect rate of habituation. Katkin and McCubbin (1969) divided 60 normal subjects into high and low autonomic activity groups on the basis of spontaneous SC activity during a rest period. Habituation to an auditory stimulus of either moderate or low intensity was tested. The high spontaneous activity group did not habituate to the more intense stimulus, though both groups did to the less intense one. Their criterion for the assessment of habituation (comparison of 5 successive blocks of 3 responses by analysis of variance)

is not easily comparable to that used by Lader, but it appears that it could not pick up a slow though statistically significant rate of habituation as efficiently as the linear regression technique. Also, their analysis includes the first response, which would elevate the mean response value in the first block of three, and thus distort the results.

The habituation of spontaneous fluctuations themselves, which was observed to occur by Lader and Wing over the 32 minute habituation period, was studied by Greene and Kimmel (1966), who found that if spontaneous fluctuations were divided into large ones (greater than 1 per cent of the subject's basal skin resistance level) and small (less than this criterion), the large ones habituated over 10 minutes while the small ones did not. A spontaneous fluctuation of any sort was defined as an increase in SC of greater than 0.1 micromho. These results were claimed to show the existence of two types of spontaneous fluctuations. However, their measure of large versus small spontaneous fluctuations does not appear to be base independent, and their criterion for a spontaneous fluctuation in mhos is not directly comparable with one based on a measure in log micromhos.

The relationship between psychological assessments of anxiety and habituation rate has also been further investigated. Koepke and Pribram (op. cit.) did not find any relationship between Taylor Manifest Anxiety Scale (TMAS) scores and speed of habituation; and Katkin and McCubbin (op. cit.) failed to find any differences between a group of high and low TMAS scorers in rate of habituation. Epstein and Fenz (1970),

arguing that the TMAS had been shown to be multifactorial in nature (Fenz and Epstein 1965, Epstein 1967, Hamilton 1959) divided their subjects into high and low scorers on anxiety subscales of Striated Muscle Tension, Autonomic Arousal and Feelings of Insecurity, as well as on the basis of the total TMAS score. They were then given ten presentations of a 115 db sound. The rate of habituation of the PGR differed in those subjects high on the Muscle Tension Scale and those low on it. They also found that groups with high and low scores on the Autonomic Arousal subscale differed in the rate of adaptation of basal SC.

Two studies have dealt with the effects of attention to the stimulus as a variable. Korn and Moyer (1968) found that the set of the subjects affected the rate of habituation (the number of trials taken to reach a criterion of 3 non-responses). Subjects who were asked to pay attention to the stimulus showed less evidence of habituation than those who were asked to relax. They also found that females habituated faster than males on the first of 2 groups of 20 tone stimuli. McDonald, Johnson and Hord (1964) compared habituation of the PGR to tones in alert and drowsy normal subjects (using an EEG criterion). Although no difference between groups was found in PGR habituation, there were differences between the groups in the SC rate. Interestingly, heart rate responses did not habituate in drowsy subjects, although they did in the alert group. These results indicate that the subject's state of consciousness cannot be ignored in assessing the results of habituation studies.

In their model for the mechanism of anxiety production, Lader and Wing (op. cit.) postulated that the habituation rate found on testing is determined at least in part by an innate habituation factor, as well as by situational variables. If this is so, then one would expect to find some intra-individual consistency in repeated tests of habituation. Thus, Montagu (1963), using a regression method identical to Lader and Wing's, found that the absolute rate of habituation (Lader's 'H' value) did not vary in successive tests. Scholander (1960) also found no habituation between sessions and a significant concordance in individuals between 'change values' of PGR within sessions (claimed as an index of habituation) over 5 daily measurements. Thus it may be concluded that habituation rate is a 'reliable' measure (in the conventional psychological sense), and repeated measures will reflect a basic individual characteristic which could be related to other psychological measures.

In summary, this technique seems to have several advantages which make it suitable for studying individual differences in anxiety and arousal. It is sensitive to drug-induced changes of mood, and is reliable. Its validity as an instrument for anxiety assessment seems to be high, though its failure to distinguish between extremes of anxiety in normal subjects (in the Katkin and McCubbin study) should be noted. An important factor to be controlled experimentally is clearly the subject's state of attention and consciousness.

1. Method

a. Stimuli

The stimuli were twenty 1000 Hz tones. These were pre-recorded on magnetic tape at random intervals of between 45 and 80 seconds. The tape began with 11 minutes of silence, followed by the 20 tones, and finishing with one minute's silence. The subjects heard the tones through a loudspeaker placed two feet behind their heads, to which the tape recorder was connected. The intensity of the tones was 100 db compared to a reference level of 0.0002 dynes/cm^2. This was measured with a Dawes sound level meter.

b. Measurement technique

The method of measurement chosen for skin resistance is described below. A constant current of 10μA was passed between two double-element lead electrodes which are shown in figure 4.1. These were originally developed by Lykken (1959). The active (smaller) electrode was placed on the distal phalanx of the right thumb, and the passive one on the right forearm. This method was chosen for the following reasons:

i) The electrodes are easy to prepare, needing only to be washed and rubbed with emery paper before application.

ii) The current of 10μA was chosen to be small enough to minimise the effects of tissue damage (Edelberg et al., 1960), but large enough to ensure that the induced voltage would be high, compared with the galvanic skin potential (which would oppose the voltage due to the skin resistance) between the two electrodes.

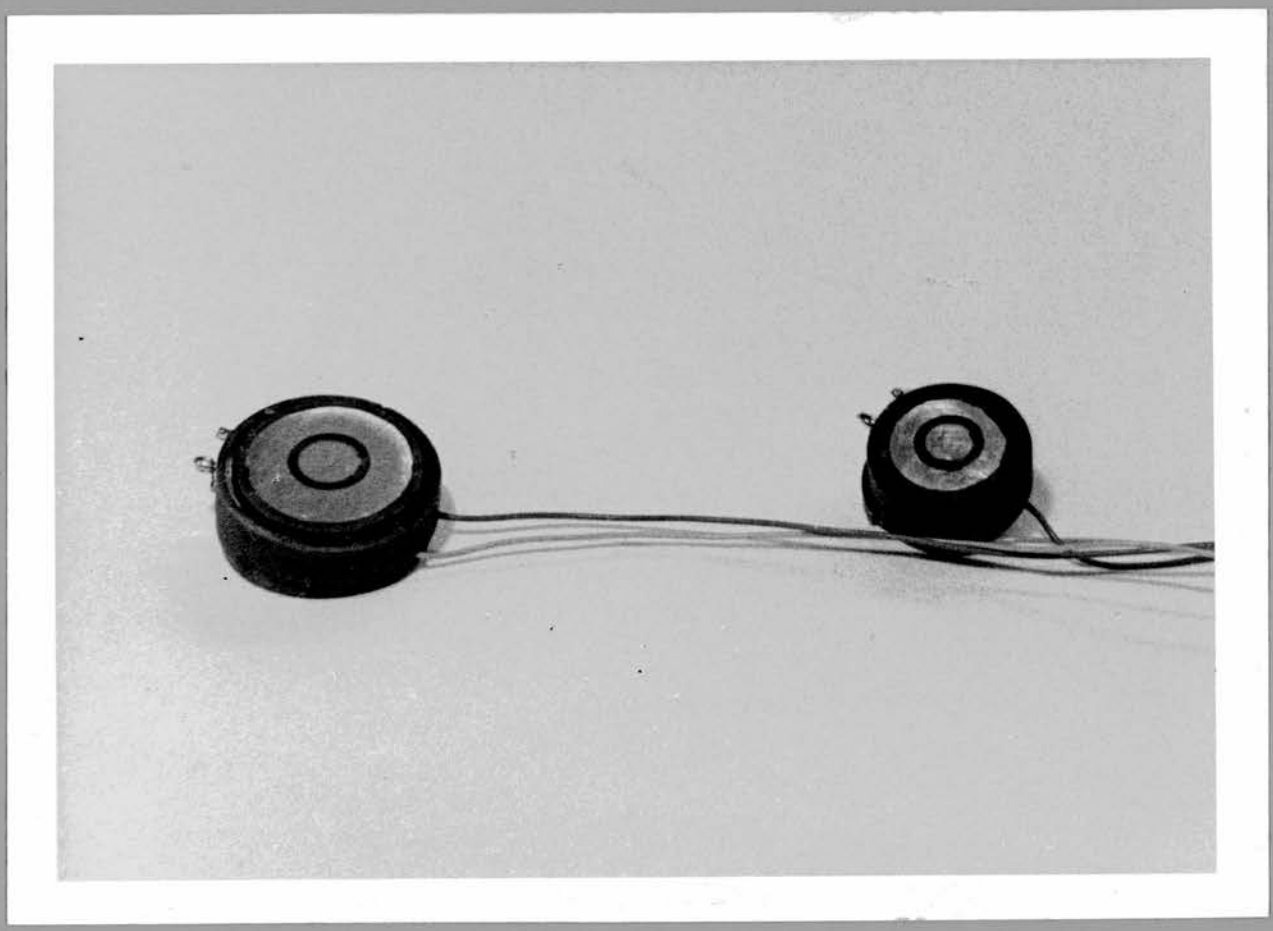

Figure 4.1: Tetrapolar lead electrodes.

The smaller electrode is attached to the thumb; the larger (earth) electrode is attached to the forearm.

The conversion of the voltage measurements to resistance units is simple.

The constant current was generated by the circuit shown in figure 4.2. This contains two separate parts. The first is the constant current generator, which consists of a 270 volt battery in series with a 27 M resistor. Compared with this, the subject's resistance is minimal (of the order of 0.5%), and changes in it will not affect the current in the circuit. The second part of the circuit is a "backing-off system". This subtracts one of several fixed voltages (0-3V in steps of 0.5V, corresponding to 0-300K), so that the signal output to the amplifier is small enough to be amplified to a high sensitivity.

The amplifier used was a Devices DC6 amplifier, which has an additional backing-off unit (0-1V). The residual signal was then recorded on one channel of a Devices 2-channel polygraph, at a sensitivity of 50mV/cm (equivalent to 5K /cm). With a knowledge of the total backing-off at any time, it was possible to work out the basal resistance from the polygraph record.

2. Procedure

The subjects were seated in the experimental cubicle and the introductory tape (see chapter 3) was played to them. The two electrode sites were then prepared. The thumb was rubbed with a piece of cotton wool and a corn plaster was applied; the forearm site was briskly rubbed until a slight erythema was produced, in order to

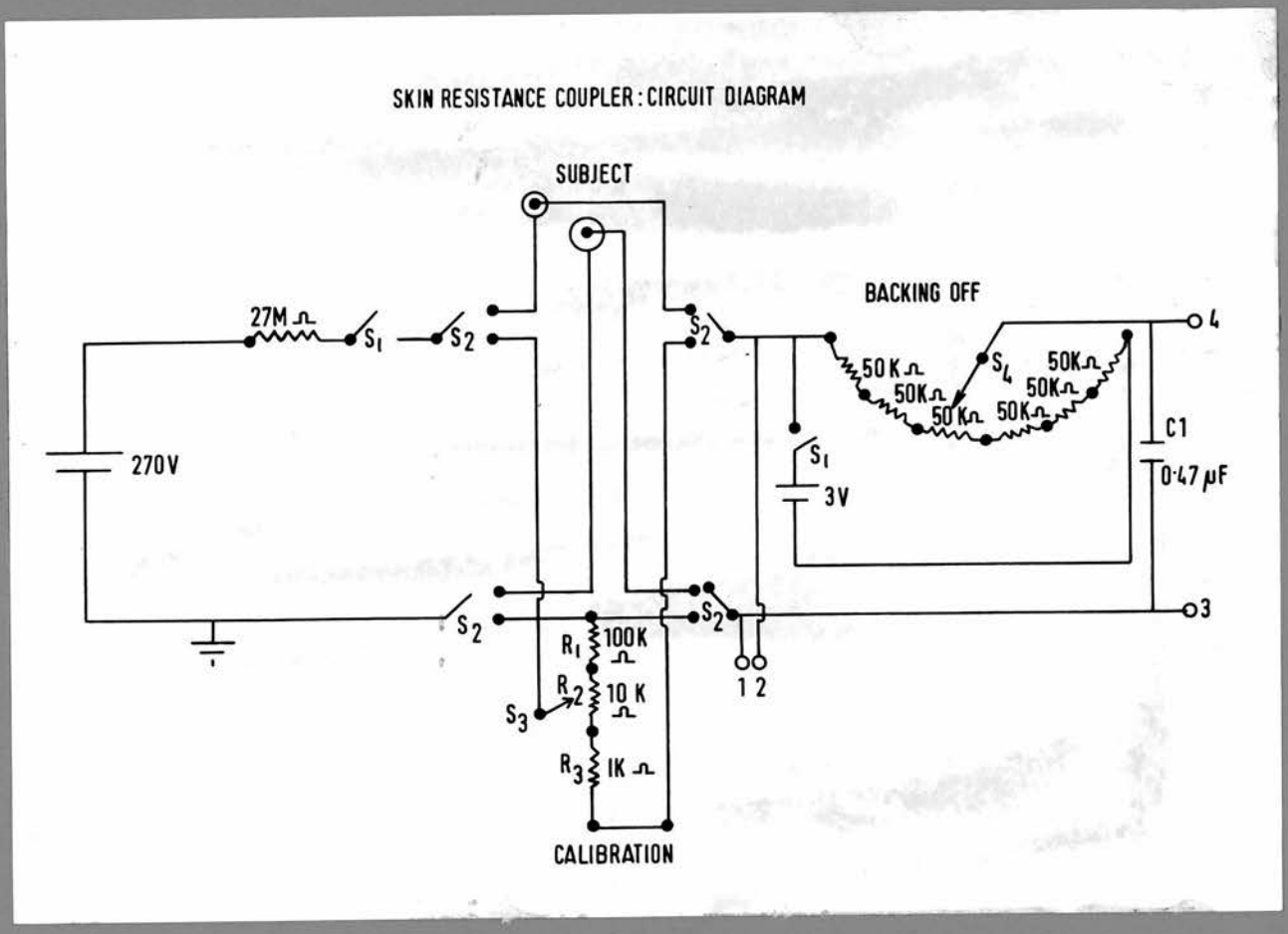

Figure 4.2: Circuit diagram of the skin resistance coupler.

KEY

S_1 main of-off switch
S_2 selects subject or calibration circuit
S_3 selects value of fixed (calibration) resistance
S_4 selects backing off voltage

Outputs 1+2: to data logger
Outputs 3+4: to DC6 amplifier
C1 smoothing capacitator.

reduce the electrical resistance at the site. The cup of the corn plaster was then filled with electrode jelly (KY jelly), as were the two electrodes, and these were applied, the small (active) electrode above the corn plaster and the large one directly to the skin. The diameter of the corn plaster was such that the current density at the active electrode was 14$\mu a/cm^2$. The electrodes were then made secure with surgical tape and plugged into the amplifier. The subject was reassured that he was in complete contact with the experimenter at all times and the door of the experimental cubicle was then closed. After the DC6 amplifier had been adjusted, the tape with the pre-recorded tones was turned on. The marker pen on the polygraph was used to record the time of presentation of each stimulus.

3. Analysis of Results

Each polygraph record was analysed in the following way. Eleven readings of skin resistance were taken, one each minute, counting backwards from the time of the first stimulus. The system for scoring the responses themselves was identical to that used by Lader and Wing (op. cit., p. 52). A final reading of resistance was taken one minute after the last stimulus.

The analysis of each subject's data was performed by a computer program, (listed in Appendix 2) which took the 52 skin resistance readings (11 basal + 2x20 stimuli + 1 basal at the end) for each subject, and converted them to log conductance units.

The choice of log conductance units as the unit of skin resistance measurement can be justified on both empirical and theoretical grounds. Lacey and Siegel (1949) and Haggard (1949) both demonstrated that the transformation of resistance readings to conductance or log conductance gave a distribution of readings close to statistical normality. More importantly, research on the peripheral physiological mechanisms of the galvanic skin response (Thomas and Korr, 1957, Lader, 1970) indicates that the electrical properties are dependent on the number of active sweat glands, which can be considered as electrical resistances in parallel. Since the conductance of a network of resistors in parallel is additive, it is the preferred unit of measurement. The transformation to log conductance allows a base-free measure of response to be computed.

Using the procedure adopted by Lader and Wing, the following measures were computed from the data:

a) the basal log conductance averaged over successive blocks of 4 minutes

b) the magnitude of the first response

c) the regression of the second to twentieth responses on the logarithm of the stimulus number. Two parameters, the 'a' and 'b' values are linked by the equation

estimated response to nth stimulus = a + b log n.

d) an analysis of variance estimating the variance associated with the regression line and the error variance. (Snedecor, 1956). The 'F' ratio tests the hypothesis that the regression coefficient obtained differs significantly from zero.

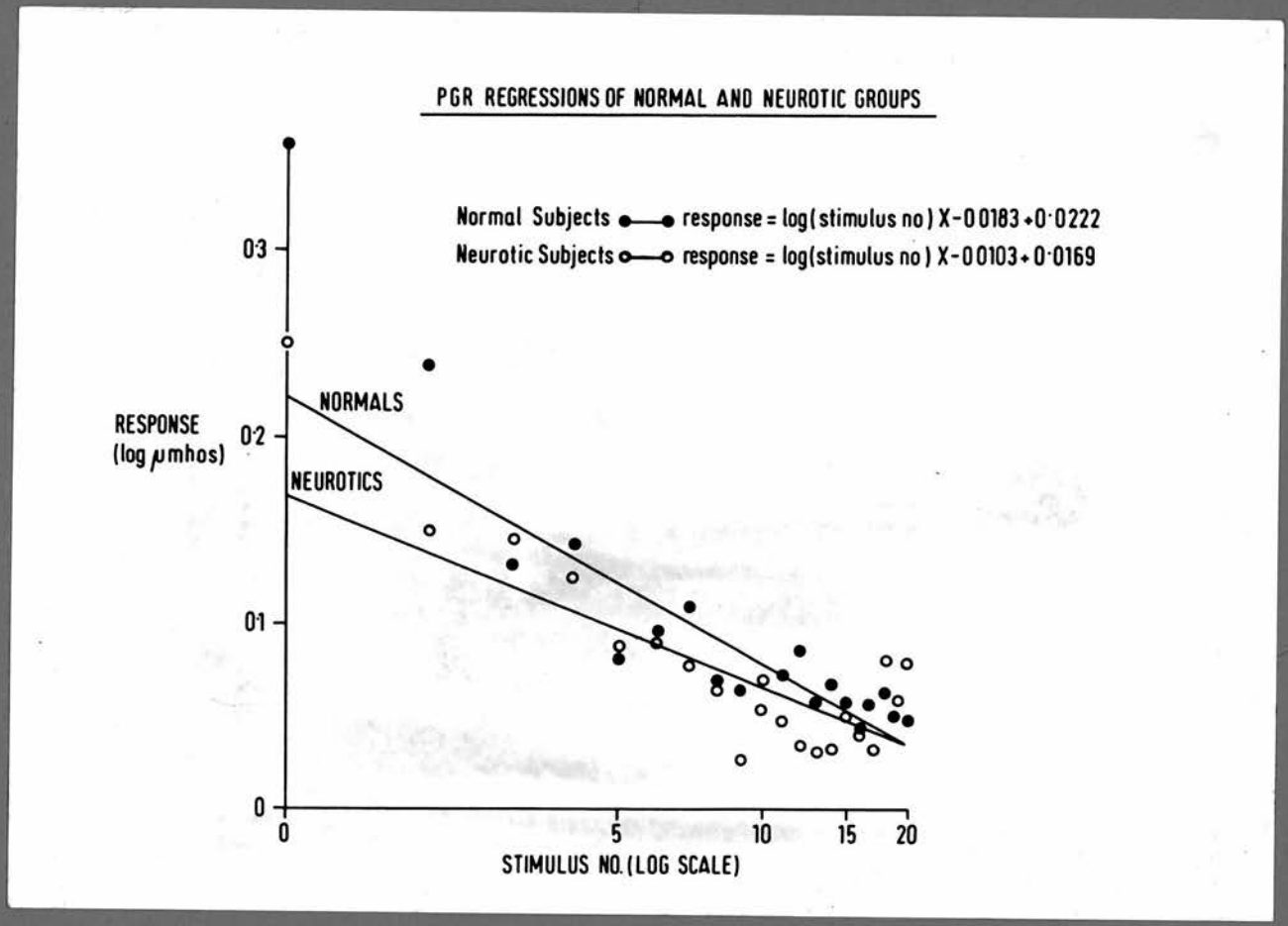

Figure 4.3: PGR regressions of normal and neurotic groups.

e) the number of SCSFs per minute was calculated by hand from the polygraph recording. Only fluctuations greater than 0.003 log umho were counted. Because this criterion corresponded to a variable change in skin resistance, which was dependent on the basal level, a table was prepared of the minimum resistance change equivalent to an SCSF over the whole range of basal levels. Thus, SCSFs could be counted quite easily.

To begin the analysis of results, the mean response curve for the normal and neurotic subjects was computed. The first, second, third, etc. readings were averaged over all subjects in each group to give two group regressions. These are shown graphically in figure 4.3.

An analysis of covariance (Snedecor, 1956) tested the hypothesis that these two regression lines differ significantly. (Table 4.1).

The results show that the difference between the slopes of the two regression lines is not quite significant at the 5% level. As expected, normal subjects habituate more rapidly than the neurotic group. However, the magnitude of the slope and the y-intercept are much lower than the values reported by Lader, even though the stimuli were supposed to be identical.

TABLE 4.1

Analysis of Covariance Comparing the Group Regression Lines

(stimuli 2-20)

Source of Variance	Σx^2	Σxy	Σy^2	df	SS	MS
Normal Group	1.47736	-0.02110	0.00039	17	0.92648	0.05449
Neurotic Group	1.47736	-0.01371	0.00019	17	0.63717	0.03748
Within Groups				34	1.56365	0.04599
Regression coefficient				1	0.18506	0.18506
Pooled Regression	2.95472	-0.03481	0.0058	35	1.74871	0.04996
Adjusted Means				1	0.30880	0.30880
Total				36	2.05751	

F = MS (regression coefficient) / MS (Within gps variance) = 4.024 ($p<0.1$).

The group means of several other measures were compared by means of a one way analysis of variance. These measures were -

a. The number of spontaneous fluctuations in the 4th to 8th minutes of rest, prior to the auditory stimuli.

b. The slope of each individual's regression (the 'b-value')

c. The intercept with the y-axis of each individual's regression (the 'a-value')

d. The skin conductance at the start of the test (the mean of the readings in minutes 1-4)

e. The skin conductance at the end of the test (the mean of the

readings in minutes 29-32)

f. The size of the first response

These results are shown in table 4.2, where it can be seen that there are no differences between any of the groups on any of these four measures. Except for the size of the first response, this is in complete contradiction to Lader and Wing's results, and indicates that the within group variance is very large.

Another way of examining the habituation data is to divide each of the groups into 'habituators' and 'non-habituators', on the basis of the statistical significance of each individual's regression line. It would be expected that the proportion of individuals who were habituators in the normal group would be greater than the proportion of the neurotic group. Table 4.3 shows the numbers in each category; a chi-square test leads to a rejection of the hypothesis that the proportion of habituators in the two groups differs. This result is also in contradiction to Lader and Wing's findings.

Following this failure to distinguish between the control groups, it did not seem worth analysing the results in further detail. Several possible reasons could be put forward for the lack of success. One is that many of our subjects were drowsy during the test. It is the author's own experience that in the inevitably unstimulating environment in the experimental room it was easy to doze off, and some of the subjects reported quite spontaneously that they had felt drowsy during the test. A modification to the experimental procedure, such as asking the subject to respond to each stimulus by pressing a button, might help

TABLE 4.2

PGR HABITUATION: GROUP RESULTS

Variable	Asthmatics n=65		Ref. Ast. n=11		Neurotics n=19		Normals n=22		Analysis of Variance
	Mean	S.D	Mean	S.D	Mean	S.D	Mean	S.D	F.Value
Spontaneous Fluctuations	6.0	6.2	8.1	11.3	8.6	7.5	7.8	5.5	0.18
'A' Value ($x10^4$)	252	242	244	364	165	205	221	229	1.41
'B' Value ($x10^4$)	-160	193	-114	207	-98	155	-131	172	0.80
SC at start (log umhos $x10^4$)	844	231	818	171	740	207	818	189	1.12
SC at end (log umhos $x10^4$)	866	203	854	257	781	166	818	144	1.02
First response (log umhos $x10^4$)	359	279	372	483	290	252	371	278	0.30

TABLE 4.3

'HABITUATORS' AND 'NON-HABITUATORS'

	Habituators	Non-Habituators	
Neurotics	9	9	18
Normals	13	9	22
	18	22	40

X^2 (with Yates' correction) = 0.07 (N.S.)

to keep subjects reasonably alert during the test. The small responses observed in all groups during this study may have been due to the position of the loudspeaker, which was about a foot further from the subject than Lader reports, with the result that the loudness of the tone would have been lower.

Another explanation for the lack of discrimination between the two groups is that the neurotic group were not similar to Lader and Wing's. Since this point is also pertinent to the results of the forearm blood flow assessment, it will be discussed in some detail at the end of this chapter. However, this cannot be an explanation for the finding that only 13 out of 22 normal subjects were 'habituators'.

C. **Forearm Blood Flow**

One of the principal physiological components of the state of psychological arousal is vasodilatation in the limb muscles. In the resting state, much of the blood flow through the limbs takes place through the skin blood vessels, but in emotional states, skin blood flow decreases, and muscle blood flow assumes an important role -- possibly to prepare the organism for "fight or flight". One consequence of this vasodilatation is a large increase in forearm blood flow (even though the vasodilatation may cause a decrease in arterial blood flow pressure and would thus be expected to reduce blood flow). This effect has been the subject of much investigation.

Barcroft *et al.* (1960) investigated the mechanisms underlying this vasodilatation. They concluded that both a humoral mechanism and a cholinergic nervous mechanism involving the sympathetic vasodilator fibres to the arm muscle could be operative.

Early studies of both skin and muscle blood flow have been reviewed by Ackyer (1956), who also reported a study [illegible] investigating the relationship of anxiety to peripheral vasomotor activity. In this, he found that non-psychiatric and non-anxious psychiatric groups of subjects could be reliably differentiated from an anxious group on the basis of the pulse volume change during sleep.

The most consistent recent set of studies on forearm blood flow have been reported by Kelly and his co-workers. Kelly (1966) found that basal forearm blood flow, measured by venous occlusion plethysmography reliably differentiated a group of 20 chronically anxious subjects from two control groups of 40 mixed neurotics and 40 normal subjects. The test-retest reliability was assessed using a sample of 20 subjects from the three groups, whose clinically assessed anxiety had not changed over a period of time (3 weeks to 10 months later). The correlation between the forearm blood flow on the two occasions was highly significant ($r > 0.90$). A fall in forearm blood flow was noted in patients whose anxiety had been treated and whose clinical state had improved as a result of the treatment.

Kelly and Walter (1968) found that the technique distinguished between different psychiatric diagnostic groups. They found that all groups showed a change of blood flow when stressed (being asked to do mental arithmetic) and that the degree of change (expressed as a percentage of the basal level) was inversely related to the basal level, non-anxious subjects showing the greatest increase. Forearm blood flow was thought to reflect the "free-floating" anxiety that was present,

since it was high in patients with chronic anxiety states and agitated depression, and low in those with monosymptomatic phobias (and, of course, normal subjects).

In another study, Kelly and Martin (1969) obtained measures of forearm blood flow, systolic and diastolic blood pressure and heart rate from groups of normal, chronically anxious and neurotic subjects. The results confirmed the 1966 and 1968 findings. A factor analysis of the psychophysiological and psychometric test data indicated that the Taylor Manifest Anxiety Scale, Eysenck Personality Inventory 'N' scale and 'E' scale (in the direction of introversion) subjective ratings of anxiety, and the physiological measures all loaded on one factor, which could reliably be identified as an anxiety-neuroticism dimension. Kelly et al. (1970) also reported good correlations between ratings of clinical anxiety and basal forearm blood flow, but not between depression ratings and forearm blood flow, although many of the patients in that study were quite severely depressed.

Both Harper et al. (1965) and Gelder and Matthews (1968) have observed that a large increase in forearm blood flow occurs when phobic subjects are asked to imagine phobic images, and this seems to confirm Kelly's impression that it is a reliable indicant of anxiety present at the time of testing.

Kelly (1966) lists a number of physical disorders which can also produce a large forearm blood flow. Clearly these would have to be considered as one possible cause of high results if the technique were to be used in a psychiatric setting. It is possible that in some

asthmatics, the presence of cor pulmonale would produce high cardiac output, leading to increased FBF which was not related to anxiety. Other factors known to influence forearm blood flow are age, which causes an increase in women (Imms and Kelly, 1966), and time of day (Kneko, et al., 1968).

1. Method

The plethysmograph used was similar to the one described by Kelly (1967), and is shown in figure 4.4. Figure 4.5 shows schematically the method of assessing volume changes. Water is free to flow up the "chimney" B. A metal tube of small diameter is connected to a side-arm of the chimney at A, and this is connected to a pressure transducer (a linear micromanometer with an A300 capsule). This measures the pressure difference between the pressure at A and atomspheric pressure, which is equal to the pressure of water in B. This is proportional to the volume in B, since the internal diameter of the chimney is of constant cross section.

The blood flow in the forearm is controlled by two blood pressure cuffs, attached to the arm on either side of the plethysmograph. The distal one was inflated throughout the period of measurement, to a pressure of 200 mm. Hg. This prevents any arterial blood flow from the arm to the hand. The proximal one is placed near the elbow, and is inflated during the period of measurement to a pressure of 60 mm. Hg. Blood can flow into the forearm but cannot leave it. The rate of increase of the volume of the forearm during this period is therefore a

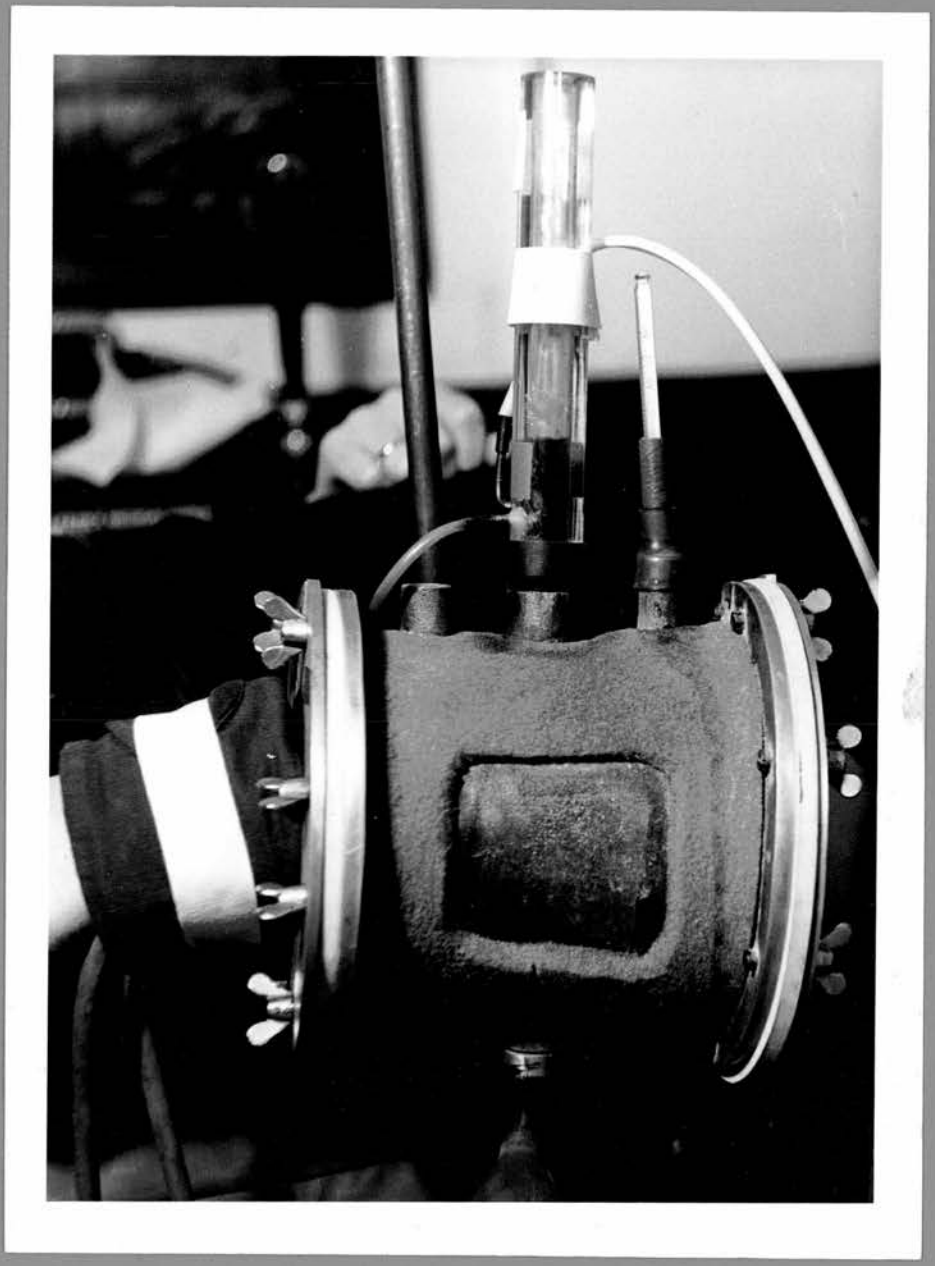

Figure 4.4: Venous occlusion plethysmograph.

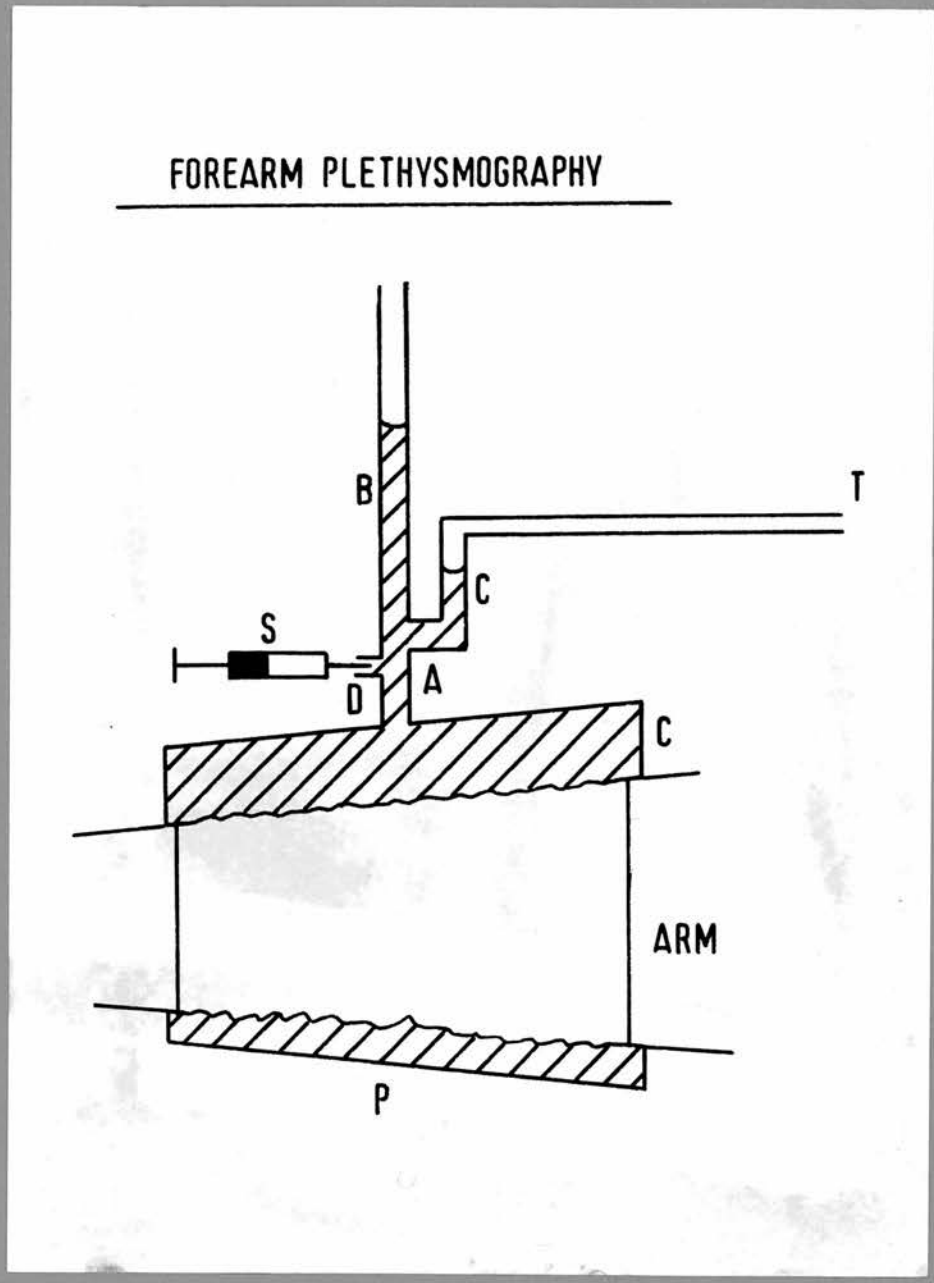

Figure 4.5: Technique of forearm plethysmography.

A = side arm for connection of pressure transducer T.

B = "chimney".

C = (stippled area) is filled with water.

D = Inlet for syringe S.

P = body of plethysmograph.

measure of the amount of blood flowing into the forearm. The inflation of the cuffs was remotely controlled from the laboratory, so as to avoid disturbing the subject while 'basal' measures were being made.

2. Procedure

a. An appropriate rubber cuff was chosen to fit the subject's left forearm.

b. The cuff and the forearm were dusted with talcum powder and the cuff placed on the forearm.

c. The subject lay on a couch with his left arm held to the side, in a position such that it was above the sternal angle.

d. The subject's arm was slipped into the plethysmograph and the rubber cuff was secured to it with wing nuts and metal plates at each end of the device.

e. The remaining space in the plethysmograph was filled with water at a temperature of 34 degrees ^{o}c., care being taken that there were no air bubbles.

f. The two sphygmomanometer cuffs were put around the subject's arm at the wrist and near the elbow (the exact position depending on the size of the individual's arm).

g. The subject was warned that the wrist cuff was about to be inflated and that it would feel "tight", and that the experiment would last only a few minutes more. The cuff was then inflated.

h. A syringe was attached to the tube D in figure 4.5, and the system was calibrated by injecting and extracting 3 mls. of water, repeated four times. The displacement of the pen on the polygraph was measured.

i. Ten measurements of forearm blood flow were made, one every 30 seconds. The elbow cuff was blown up for about 15 seconds and released for the remainder of the 30 second interval.

j. The calibration was repeated after the measurements.

k. The water in the plethysmograph was run off. An estimate of the subject's arm volume was obtained by subtracting the volume of the water from the known total volume of the plethysmograph.

3. Computation of results

As the blood flows into the forearm, the volume increases steadily and thus should give a smoothly rising slope on the polygraph record. For each slope, a line of best fit was drawn by the eye, and the angle it made with the horizontal was measured. It can be shown by simple trigonometry, that

$F = S \tan A/c$

where

F = forearm blood flow in ml./min.

S = paper speed of the polygraph in cm./min.

C = calibration constant (cm. of polygraph displacement per cc. of water injected)

A = angle of slope.

Any readings observed to be abnormally low in relation to the rest of that subject's readings were eliminated. The mean of the remaining readings was taken as the measure of "basal" blood flow. Kelly used the lowest three, but since there were a considerable number of artefactually low readings, it seemed more advisable to incorporate all valid readings. The reading was then divided by the subject's arm volume to obtain reading ml./min./100 ml. forearm.

4. Results and discussion

Table 4.4 shows the mean basal forearm blood flows of the four groups. It can be seen that these are all similar, and analysis of variance revealed no differences between the groups. The correlations between Taylor Manifest Anxiety Scale score and forearm blood flow within each of the four groups are set out in table 4.5. None of them are significant, and all show a negative association -- which is completely contrary to other published results, and to prediction.

TABLE 4.4

BASAL FOREARM BLOOD FLOW

Group	Mean	s.d.	n
Asthmatics	1.91	1.12	65
Ref. Ast.	2.34	1.15	11
Normals	1.69	0.98	22
Neurotics	1.89	1.34	20

ANALYSIS OF VARIANCE

Source	df	SS	MS	F
Between Groups	3	3.104	1.035	0.80 (N.S)
Within Groups	112	145.311	1.297	
TOTAL	115			

TABLE 4.5

PRODUCT-MOMENT CORRELATIONS BETWEEN TMAS AND FBF

Group	n	r
Asthmatics	63	-0.03
Ref. Ast.	11	-0.45
Neurotics	19	-0.09
Normals	22	-0.27

All correlations are non-significant.

The failure to replicate Kelly's series of findings can be explained by one or both of two factors. The procedure could in some way have led to artefactual results, or the neurotic group of subjects were dissimilar to those of previous studies.

The criteria by which our neurotic group were selected were noted in the previous chapter, and it can be seen that the emphasis in selection was placed on the presence of anxiety symptoms. Yet the group's TMAS scores were ten points lower than those reported by Kelly and Martin (1969) for a group of individuals with chronic anxiety states, and were perhaps more similar to those of the psychoneurotic group in that study, whose forearm blood flow did not differ significantly from that of their normal subjects.

The non-significant and consistently negative correlations found between TMAS scores and forearm blood flow cannot be explained by the postulation of sampling deficiencies, however. Even if no significant differences between groups were obtained, it should have been possible to obtain positive associations between questionnaire and physiological measurements within each group - especially in the very heterogeneous asthmatic group. One can only conclude that "free-floating" anxiety was n present in many of our subjects, or that the TMAS is not a suitable measure of it.

The other possible explanation for the results is a deficiency in the method used. That this cannot be ruled out is indicated by the following observations.

a. The mean value for all the four groups was much lower than comparable figures reported by Kelly et al. (1968, 1970, op cit).

b. The correlation between the "calibration factor", c, and blood flow was significant and negative. If it is assumed that the c varies randomly, then no relationship should be expected. This is a very strong indication that there is a deficiency in the method used for measurement of volume change.

D. General Discussion of the Methods

Both of the anxiety assessment techniques have signally failed to discriminate between criterion groups of normal and neurotic patients. Since special attention was paid to the selection of a group of appropriately anxious subjects, one is bound to ask why these results have been obtained. Several possible reasons, relevant to both of these techniques spring to mind.

1. Procedural deficiencies

It was noted above that there is reason to suspect the reliability of the method used to assess volume change in the forearm blood flow measurements. This is not likely to be a complete explanation for the lack of discrimination between the groups, since both in this study, and in others in the same laboratory (Morakinyo, unpublished data) a wide range of readings has been obtained, i.e. the equipment used is perfectly capable of registering high readings. Similarly, the

difficulties encountered in the PGR habituation procedure may have contributed markedly to the indeterminate results obtained. Since both tests gave results indicating that the group of subjects selected on the basis of being anxious were not revealing this in these tests, then it is also possible that they differ in some critical way from the populations sampled in previous studies, and this is discussed in the next section.

2. The characteristics of the neurotic group of patients

Lader and Wing (op cit., p.79) describe the selection of their 20 anxious patients in some detail. They were all 'known to be suffering from anxiety states, ... complained of symptoms referable to the autonomic nervous system and the muscular skeletal system. Typical symptoms were palpitations, sweating, especially of the hands, muscle tension, dry mouth, vomiting and diarrhoea. Patients whose anxiety was clearly secondary to other illnesses (such as the psychoses) were excluded). They were all judged to be showing "situational" or "free-floating" anxiety. Kelly et al. (1969, 1970) do not make the criteria for inclusion in their group of anxiety state subjects so explicit, but the patients they studied seem to be similar.

In contrast, the criteria adopted for this study of "patients treated in hospital for a psychoneurotic disorder characterised by anxiety" may have led us to incorporate patients whose anxiety was not primarily somatic. Furthermore, because the anxious patients were not of primary importance in this study, and were to be yoked to the asthmatics on the basis of age and sex, they were obtained by consulting medical records of recent patients at the Royal Edinburgh

Hospital, rather than by asking a consecutive series of referred patients to participate. It was therefore necessary to rely on written case notes and diagnoses which were made by a variety of psychiatrists rather than by the ones involved in the research. The subsidiary diagnoses of immature personality, reactive depression, personality disorder which were made for many of these patients lead one to suspect that these patients are not clinically equivalent to those tested in the original demonstrations of these techniques. They were probably "trait" anxious individuals in contrast to those in the earlier studies. This is confirmed in part by the observer ratings of personality traits made by the psychiatrists of these individuals.

3. Drug effects

One of the notable features of the PGR habituation and forearm blood flow techniques is their sensitivity to the effects of psychotropic drugs. The PGR habituation technique has been used as a bioassay method for the comparison of the effects of chlordiazepoxide and amylobarbitone sodium (Lader and Wing, op cit.). Thus, our anxious patients were asked to discontinue the use of any anxiolytic drugs they had been prescribed for a period of 48 hours before attending the laboratory. However there is no way of confirming that all of them in fact cooperated in this matter, and one could surmise that several did not. Furthermore, there is suggestive evidence (Lewis, 1969) that the withdrawal effects of certain tricyclic drugs and sedatives may last for several weeks, as judged by

the disruption of normal sleep patterns. This presumably reflects a disruption of CNS functioning. There is no direct evidence that the drugs in common clinical usage for the control of anxiety symptoms would have similar effects, nor that CNS effects would necessarily be accompanied by changes in autonomic nervous system functioning; however, the possibility cannot be ignored.

4. What aspects of anxiety do these physiological measures reflect?

Lader and Wing concluded that the physiological measures were a reflection of overt anxiety, since they correlated well with observer ratings of this quantity made immediately before the physiological recording. It was noted in the review of relevant studies that few relationships had been found between the scores on the Taylor Manifest Anxiety Scale and measures of PGR habituation, and this was confirmed in the findings reported here. Kelly (1966) claims that high FBF is one of the signs of anxiety, but he notes that scales such as the TMAS reflect the symptoms present, and may also be a measure of neuroticism. What is equally likely is that the scale may also be measuring anxiety-proneness, since the form of many of the questions is 'I frequently feel ....'. This is a dispositional measure, and, if this is the case, then it is not so surprising that low correlations were obtained between psychological and physiological measures. For individuals who are currently anxious will both show physiological evidence of this state and report anxiety symptoms on the TMAS. However, high anxiety scale scores will not necessarily be associated with physiological hyper-reactivity, if the anxiety scale is measuring a mixture of anxiety symptoms and anxiety-proneness.

5. The use of physiological measures of anxiety

The findings reported in this chapter have shown some of the pitfalls of these two techniques. Given the expense in time and practical difficulties associated with these procedures, they do not yet appear to be useful additions to the battery of techniques available for psychological assessment. Without a detailed formulation of the type of anxiety that these measures reflect, and a convincing demonstration of the validity and reliability of the methods (such as would be demanded of any new psychological test), they will add only confusion to research and individual assessment. Even if the tests could be shown to be valid, the wide differences that exist between individuals would only make them useful as measures of individual change, induced (for example) by drugs or other psychiatric treatment.

CHAPTER 5

PSYCHOMETRIC TESTS OF PERSONALITY AND ANXIETY

A. Introduction

The tests used in this phase of the research were chosen because they fulfilled the following important criteria:

a) they were all standardized objective instruments which had been well documented.

b) The theoretical basis of each of the tests had been explicitly formulated by their authors, and estimates of their reliability and validity were available.

c) They had all previously been shown to distinguish between various psychiatric groups and normal control subjects.

d) They were straightforward to administer and did not take too long to complete.

Most importantly, they measured personality dimensions for which, as was noted in the review of literature, there was some reason to expect that asthmatics would differ from the normal and neurotic groups; also, some of these dimensions may have a primarily biologically determined basis and are thus of especial interest

It would not be appropriate to attempt to summarize the vast literature on extraneous factors influencing test-results, such as response sets, except to say that findings in this field have often been conflicting and unclear. There is no clear evidence that psychiatric patients (as distinct from college students) are

not co-operative and truthful in a test situation, especially where there is nothing to be gained by dissimulating. Thus one can assume that the possible effects of response sets are not likely to affect to any large extent the test results reported here.

The four tests used were the Eysenck Personality Inventory, the Hostility and Direction of Hostility Questionnaire, the Taylor Manifest Anxiety Scale, and the Cattell 16 Personality Factor Questionnaire. They are all described in Section B below, and copies of the tests can be found in Appendix 1.

B. Description of the tests

1. The Eysenck Personality Inventory (EPI)

This test (Eysenck and Eysenck, 1964) was developed from its predecessor, the Maudsley Personality Inventory (MPI) (Eysenck, 1959). The personality theory underlying these tests has already been mentioned in Chapter 2 and an account of the origins of the MPI is given by Eysenck and Eysenck (1969). The test contains three scales, Neuroticism (N), Extraversion (E) and a Lie scale. The Lie scale as a measure of the possibility of a subject 'faking good' on the test; a high score on this scale is said to be cause for regarding the N and E scores of that subject with some scepticism. However, Knowles and Kreitman (1964) oppose this use of the Lie scale, since they found a significant correlation between the Lie and Neuroticism Scales.

The reliability of both N and E scales is good; the test-retest reliability with normal subjects for both scales is over 0.80 and the split-half reliabilities are similar (Eysenck and Eysenck, 1964). However, the temporal stability of the test, which measures what are said to be stable traits of temperament, has been less clearly demonstrated. Lunghi and Ryle (1969) report test-retest correlations of only between 0.54 and 0.67 for groups of patients and healthy university students; they concluded that either neuroticism and extraversion are less stable traits than is usually claimed or that the test is an unsatisfactory measure of these traits. However, an equally possible explanation is that students may be undergoing marked psychological changes under the influences of a university environment and are thus not the best people on whom to establish the reliability of psychological tests over time.

Ingham (1966) studied the changes in MPI scores in neurotic patients over three years. He concluded that the variations found in both neuroticism and extraversion scores probably arose from causes associated with the occurrence of the psychiatric disorders and its subsequent treatment. Thus, one should conclude on the basis of both of these studies that the long-term stability of these traits is not as great as the theories of its author would lead us to believe.

2. The Hostility and Direction of Hostility Questionnaire (HDHQ)

This test (Caine, Foulds & Hope, 1967) was designed to measure a variety of possible manifestations of hostility. The rationale behind the test and the nature of its subscales has been described by Foulds and his colleagues (Foulds 1965, Foulds, Caine & Creasy, 1960). The test consists of a selection of questions from the MMPI, and scores on five subscales can be derived from it. These are Acted Out Hostility (AH), Criticism of Others (CO), Projected or Delusional Hostility (PH), Self-Criticism (SC), and Guilt (G). Of these five subscales the first three were said to be a measure of extrapunitiveness (hostility directed outwards), while the latter two were said to measure intropunitiveness (hostility directed inwards against the self). Foulds, et al (op. cit.), found that all the correlations between the subscales were positive, allowing them to think of hostility as a unitary concept. Hope (1963) performed a principal components analysis of matrices of subscale intercorrelations derived from data on normal and neurotic groups. He confirmed that the first component was unipolar with all five tests represented, and observed that the second contrasted the intropunitive subscales SC and G with the extrapunitive ones, AH, CO and PH and could therefore be interpreted as a direction of hostility factor. From this, he derived easily calculable component scores; for Total Hostility the formula was AH + CO + PH + SC + G, and for Direction of Hostility the formula was (2SC + G) - (AH + CO + PH); thus, positive scores are equivalent to intropunitiveness.

The Manual of the Hostility and Direction of Hostility Questionnaire (Caine, et al., op. cit.) includes details of the reliability and validity of the HDHQ, together with details of how to administer and score the test. On a sample of 30 normals, the test-retest reliability for total hostility was 0.75, while that of Direction of Hostility was 0.51. On testing a further 64 normal subjects, Philip (unpublished data) found coefficients of reliability of 0.64 for Total Hostility and 0.69 for Direction of Hostility.

Norms for two different populations of normals and neurotics have been summarized by Philip (1968). He found that the two components Total Hostility and Direction of Hostility could be established in two different samples of normals and neurotics, one from South East England (Hope's original data) and the other from Aberdeen. However, Aberdeen normal subjects score more highly on both Total Hostility and Direction of Hostility than the English sample, and it is not known whether these differences are due to sampling or to real regional (perhaps cultural) differences. In any comparisons made with published norm in this thesis, the Aberdeen figures will be used, as they are based on a larger number of subjects who would probably ethnographically resemble the subjects in this study more closely than Hope's English group.

There have been several studies using the HDHQ to investigate hostility in various psychiatric groups. The HDHQ Manual (op. cit.) quotes results for various criterion groups used to establish the validity of the test. Neurotics score higher than normals on

Total Hostility and Direction of Hostility, while psychotics score higher than both groups. Foulds (1966) gave both the test and the Symptom-Sign Inventory (Foulds, 1965) to a large number of psychiatric patients in several diagnostic groups. He found that those patients in almost all groups who complained of predominantly 'psychic' symptoms scored higher on Hostility and Direction of Hostility (i.e. more intropunitively) that those whose complaints were primarily 'somatic'. He suggested that somatization of symptoms might be a substitute form of intropunitiveness, a conclusion in agreement with the psychoanalytic concept of the conversion reaction. This result has clear implications for interpreting the results of patients with bronchial asthma.

3. Taylor Manifest Anxiety Scale (TMAS)

In contrast to the EPI Neuroticism Scale, which claims to measure a fundamental dimension of personality, the TMAS was constructed to measure the overt or manifest symptoms of anxiety. It was originally used to assess anxiety in studies of the effects of drive level on conditioning. The test is described by Taylor (1953). In its original form, it consisted of 50 items from the MMPI selected

by clinicians as describing the symptoms of chronic anxiety, together with 135 "buffer" items. Normative data is given by Taylor (op. cit.) for 1971 male and female students, 683 airmen and 103 neurotic and psychotic patients, whose median score is at the 99th percentile of the normal subjects.

The test-retest reliabilities reported by Taylor are in the region of 0.8, which is high, bearing in mind that the test is allegedly sensitive enough to be able to detect small changes in anxiety. Indeed, it is not made clear by Taylor how much she considers the test to measure state anxiety, or whether it is more a measure of anxiety as a characterological trait. Kelly, (1966) feels that the test is a good measure of clinical anxiety, and Lader and Wing (1966) suggest that it measures the subject's awareness of anxiety and his reaction to it when it is present.

Fenz and Epstein (1965) have queried the unifactorial nature of manifest anxiety by constructing three subscales of manifest anxiety, measuring striated muscle tension, autonomic arousal, and feelings of fear and insecurity, respectively. A factor analysis of students' results on these tests showed a general

factor of anxiety on all three subscales, and a specific factor associated with muscle tension; by virtue of its association with feelings of hostility, they felt that this factor was related to an "outward" expression of anxiety, while autonomic arousal corresponded more to an "inward" expression of energy. The conceptual distinction between autonomic arousal and muscle tension has also been made by other workers (Hamilton, 1959; Buss, 1962). Crumpton, et al., (1967) have also confirmed that in psychiatric patients, manifest anxiety scales principally measure subjectively physically felt anxiety, with less emphasis on uncertainty. It was also found to be related to subjective feelings of depression.

Judging from a perusal of papers where the items in a Manifest Anxiety Scale are given (Taylor, 1953; Fenz, 1965) there is no fixed form of the scale. The version used in this study was one which had been used by Kelly and Walter (1969) in a study of the relationship of forearm blood flow to acute anxiety in normal subjects and groups of psychiatric patients. Because the normative data was obtained from English adults, this seemed the most suitable version to use.

4. The Sixteen Personality Factor Questionnaire (16PF)

This test (Cattell, et al., 1970) is based on Cattell's prolific work and theorizing about personality, of which a succint account is given in The Scientific Analysis of Personality (Cattell, 1964). In contrast to the two major personality dimensions of neuroticism and introversion-extraversion measured by the EPI, this test measures 16 first-order factors, reflecting so-called

independent "source traits", and four second-order factors (two of which are anxiety and introversion-extraversion).

Brief descriptions of the factors are given below.

First Order Factors

Factor	Low Scores	High Scores
A	Aloof, cold	Warm, sociable
B	Dull	Intelligent
C	Unstable	Calm
E	Mild, submissive	Dominant, assertive
F	Serious, sober	Enthusiastic, cheerful
G	Casual, unpredictable	Conscientious, responsible
H	Shy, timid	Adventurous, 'thick skinned'
I	Tough, realistic	Sensitive, subjective
L	Trusting, adaptable	Jealous, suspicious
M	Practical, realistic	Imaginative
N	Forthright, naive	Shrewd, sophisticated
O	Self-confident, placid	Guilt prone, timid
Q_1	Conservative	Radical, critical
Q_2	Group dependent	Self-sufficient
Q_3	Uncontrolled	Controlled
Q_4	Relaxed, unfrustrated	Tense, excitable

The four Second-Order Factors are:

I Low Anxiety vs. High Anxiety

II Introversion vs. Extraversion

III Tenderminded Emotionality vs. Alert Poise

IV Subduedness vs. Independence

The scores on these factors are calculated using the following weighted combinations of the scores on the primary factors:

$$I = 0.2\ L + 0.3\ O + 0.4\ Q_4 - 0.2\ C - 0.2\ H - 0.2\ Q_3 + 3.8$$

$$II = 0.2\ A + 0.3\ E + 0.4\ F + 0.5\ H - 0.2\ Q_2 - 1.1$$

$$III = 0.2\ C + 0.2\ E + 0.2\ F + 0.2\ N - 0.4\ A - 0.6\ I - 0.2\ M + 7.7$$

$$IV = 0.4\ E + 0.3\ M + 0.4\ Q_1 + 0.4\ Q_2 - 0.3\ A - 0.2\ G$$

The test exists in several forms, two full versions, A and B, and three shorter ones, C, D, and E. Form B was used in this study.

To permit direct comparison of a person's scores on one factor with those on another factor, the raw scores on each are converted to sten scores. This is a standardising technique which uses ten equally-spaced points to cover the possible range of scores. A score of 5.5 corresponds to the population mean and scores of 5 and 6 represent $\frac{1}{2}$ standard deviation above and below the mean respectively. Scores of less than 4 or more than 7 are thus deviant, and the extreme sten scores of 1 or 10 correspond to a score at least 2 standard deviations from the population mean. Cattell (1962) provides tables giving sten equivalents of raw test scores for men and women separately. The Handbook for the test (Cattell et al., 1970)

gives details of the test construction, administration, scoring and the reliability of the first-order factors. The split-half reliabilities (form A versus form B) range from 0.71 to 0.93, and the construct validities (i.e., the extent to which the questions comprising each factor each correlate with the total factor score) range from 0.73 to 0.96, which is typical for psychometric tests of this sort.

C. Administration of the tests

The tests were administered in the standard way. The subject read the instructions for each test in the presence of the experimenter who made certain that they were fully understood. The subjects were then left to complete the tests by themselves. When the tests were complete, the experimenter checked that no questions had been omitted.

D. Analysis of results

1. Comparison of groups within the study

The results for the four groups were compared by one-way analysis of variance with unequal numbers in the groups (Winer, 1962). For any test where the F-ratio of between groups/within groups variance was significant at $p \leq 0.05$, the individual means were compared by the Tukey 'A' test (Winer, op. cit., p 89). This test is recommended in the situation where the groups contain unequal numbers, since it is a "conservative" test; i.e. it will give few type I errors (indicating a difference where it does not exist), but at the expense of missing possibly significant differences.

For each comparison of means, the studentised range statistic, q, is computed from the formula

$$q = T_1 - T_s \Big/ \sqrt{MS_e / n_h}$$

where T_1, T_s are the two means to be compared

MS_e is the error mean square term in the analysis of variance

n_h is the harmonic mean of the number of subjects in each group,

If q exceeds a critical value (obtained from tables), then the difference between T_1 and T_s is significant at the probability level associated with the critical value. This value depends on the degrees of freedom associated with MS_e and also on the total number of groups (in this case 4) from which the two are selected. The conservative nature of the Tukey test derives from the fact that an alternative interpretation of the significance of q can be made (with the Duncan Range test) which depends not on the total number of groups, but on the number of steps (in terms of rank order) between the two means. This is a more sensitive, but more error-prone test.

Winer provides tables of values of q for different degrees of freedom and different probabilities of significance. The value of q with 3 and 120 degrees of freedom that is significant at the $p=0.05$ level is 3.69, which is the value used in all the tests in the chapter.

Since the value of q is fixed in any one test of differences between means, it is convenient to compute the honestly significant difference (HSD) from the formula

$$HSD = q\sqrt{MS_e / n_h}$$

A difference between two means greater than HSD would thus be significant at the 5% level.

In the tables showing the tests on ordered means (Tables 5.2, 5.4, 5.6, 5.11, 5.13, 5.18), matrices of the differences between the group means are shown. Significant differences are marked with an asterisk.

a. Taylor Manifest Anxiety Scale

The group results are shown in table 5.1. The differences between the groups are highly significant ($F = 14.1$, $p < 0.001$). Tests on the ordered means are set out in table 5.2. It can be seen that the normal subjects and asthmatics do not differ in their scores; but both of these groups score significantly lower than the neurotics. The referred asthmatic group differs from the normal group.

TABLE 5.1

Taylor Manifest Anxiety Scale : Group results

Asthmatics		Referred Asthmatics		Neurotics		Normals		Analysis of variance
n=68		n=13		n=22		n=22		
mean	s.d	mean	s.d	mean	s.d	mean	s.d	F ratio
15.9	8.0	10.6	7.6	28.4	9.4	9.6	7.6	14.1***

TABLE 5.2

Taylor Manifest Anxiety Scale : Tests of differences between means

HSD = 7.95 MSe = 101.70

	Asthmatics	Ref. Ast.	Neurotics
Normals	~~6.4~~6.3	~~1~~1.0*	18.8*
Asthmatics		~~4.7~~5.3	12.5*
Ref. Ast.			7.8

b. Eysenck Personality Inventory

The results are shown in table 5.3. The only significant differences between groups occur on the neuroticism scale and the tests on ordered means for this scale are given in table 5.4. Here, all groups score significantly higher than the normal group, and the asthmatics are also distinct from the neurotic group.

TABLE 5.3

Eysenck Personality Inventory : Group results

Scale	Asthmatics		Referred Asthmatics		Neurotics		Normals		Analysis of variance
	n=68		n=14		n=22		n=22		
	mean	s.d	mean	s.d	mean	s.d	mean	s.d	F ratio
Neuroticism	11.4	5.4	12.2	5.7	16.2	4.2	6.3	4.5	10.50***
Extraversion	10.5	4.1	10.1	4.7	11.1	3.6	9.8	3.0	0.57
Lie	3.8	1.8	4.9	2.2	3.2	1.8	3.5	2.0	2.30

TABLE 5.4

EPI Neuroticism Scale : Tests of differences between means

HSD = 4.7 MSe = 35.01

	Asthmatics	Ref. Ast.	Neurotics
~~Normals~~	5.1 *	5.9 *	9.9*
~~Asthmatics~~	-	0.8	4.8*
~~Ref. Ast.~~		-	4.0

c. Hostility and Direction of Hostility Questionnaire

The group results are set out in table 5.5. All the subscales and the total hostility score (but not the direction of hostility score) show significant differences between groups. Where the differences are significant the tests on the ordered means are presented in tables 5.6 - 5.11.

TABLE 5.5

Hostility and Direction of Hostility Questionnaire : Group results

Scale	Asthmatics		Referred Asthmatics		Neurotics		Normals		Analysis of variance
	n=68		n=14		n=22		n=22		
	mean	s.d	mean	s.d	mean	s.d	mean	s.d	F ratio
AH	4.1	2.1	4.6	2.9	5.8	2.2	3.5	2.1	4.41**
CO	4.3	2.4	5.3	2.8	5.8	2.9	3.0	2.5	4.25**
PH	0.9	1.3	1.6	2.1	1.7	1.7	0.5	7.4	3.54*
SC	5.1	2.7	4.3	2.9	7.1	2.7	3.5	2.3	6.19***
G	1.8	1.3	2.7	1.8	4.0	2.1	0.8	1.3	12.35***
Total	16.2	6.7	18.5	9.5	24.5	9.0	11.4	6.8	9.17***
Direction	2.8	6.1	-0.4	7.3	4.9	8.0	0.5	5.0	2.43

TABLE 5.6

HDHQ - Acted on the hostility (AH) : Tests of differences between means

HSD = 1.9 MSe = 5.39

	Asthmatics	Ref. Ast.	Neurotics
Normals	0.6	1.1	2.3*
Asthmatics	-	0.5	1.7
Ref. Ast.		-	1.2

TABLE 5.7

HDHQ - Criticism of others (CO) : Tests of differences between means

HSD = 2.1 MSe = 7.24

	Asthmatics	Ref. Ast.	Neurotics
Normals	1.3	2.3*	2.8*
Asthmatics	-	1.0	1.5
Ref. Ast.		-	0.5

TABLE 5.8

HDHQ - Projected Hostility (PH) : Tests of differences between means

HSD = 1.1 MSe = 2.01

	Asthmatics	Ref. Ast.	Neurotics
Normals	0.4	1.1*	1.2*
Asthmatics	-	0.7	0.8
Ref. Ast.		-	0.1

TABLE 5.9

HDHQ - Self Criticism (SC) : Tests of differences between means

HSD = 2.3 MSe = 8.43

	Ref. Ast.	Asthmatics	Neurotics
Normals	0.8	1.6	3.6*
Ref. Ast.		0.8	2.8*
Asthmatics			2.0

TABLE 5.10

HDHQ - Guilt (G) : Tests of differences between means

HSD = 1.5 MSe = 3.38

	Asthmatics	Ref. Ast.	Neurotics
Normals	1.0	1.9*	3.2*
Asthmatics	-	0.9*	2.2*
Ref. Ast.		-	1.3

TABLE 5.11

HDHQ - Total Hostility : Tests of differences between means

HSD = 6.8 MSe = 72.03

	Asthmatics	Ref. Ast.	Neurotics
Normals	4.8	7.1*	13.1*
Asthmatics	-	2.3	8.3*
Ref. Ast.		-	6.0

As would be expected the neurotic group differs from the normal group, in all cases, and the referred asthmatic group differs from the normals on the CO, PH, SC, G scales and total hostility score. The only scale to show a difference between the normals and the asthmatics is the self-criticism scale. Also, the random asthmatics score significantly lower than the neurotic group on the Guilt Scale and the "referred" asthmatics lower on the self-criticism scale. However, there are no differences between the asthmatics and the other groups on the the three extrapunitive scales. Assuming that asthmatic's symptoms are primarily somatic, this finding accords with Foulds' view (1966, op. cit.) that somatic symptoms could be a substitute form of intropunitiveness.

d. 16PF Questionnaire

i. First order factors

The group results for the first-order factors are shown in table 5.12. The analyses of variance indicate significant differences between groups on factors A, B, C, L, O, and Q_4. The tests on the ordered means for these factors are shown in tables 5.13 - 5.18.

Although the analyses of variance for factor A gives an F value = 2.69 ($p < 0.05$), none of the differences between groups are in fact significant. This is probably due to the "conservative" nature of the Tukey 'A' test, which has been mentioned previously. With the exception of the differences on scales relating to anxiety

TABLE 5.12

16PF Questionnaire - First Order Factors : Group results

Scale	Asthmatics		Referred Asthmatics		Neurotics		Normals		Analysis of variance
	n=67		n=14		n=20		n=22		
	mean	s.d	mean	s.d	mean	s.d	mean	s.d	F ratio
A	4.9	1.6	5.5	1.8	5.5	1.4	4.5	1.7	2.69*
B	7.8	1.9	6.8	2.5	7.0	2.2	8.7	1.5	3.04*
C	5.1	1.9	4.4	2.2	3.1	1.7	5.9	2.1	6.91***
E	4.7	1.9	5.1	2.2	6.2	2.0	5.0	2.0	1.84
F	5.6	2.1	5.1	2.4	5.2	2.1	5.7	2.0	0.05
G	5.0	2.0	4.6	1.9	4.9	2.5	5.2	1.7	0.07
H	4.9	2.1	5.1	2.8	4.4	2.4	5.5	1.9	1.26
I	4.6	1.8	4.3	2.0	4.8	2.0	5.0	1.7	0.35
L	5.6	2.3	5.3	2.1	7.1	1.9	4.8	2.1	3.77*
M	5.5	2.1	5.6	2.3	6.3	1.3	5.8	1.8	1.08
N	4.7	1.8	5.1	1.9	5.4	2.1	5.1	1.7	1.05
O	5.5	1.9	6.3	2.4	7.9	2.3	5.1	2.2	8.20***
Q_1	6.7	1.9	7.1	1.6	6.7	2.1	7.0	2.0	1.13
Q_2	5.7	2.1	6.2	2.3	5.2	2.1	6.0	1.5	1.54
Q_3	5.1	2.0	5.4	2.8	4.8	1.9	6.1	1.9	2.02
Q_4	5.9	1.8	6.1	2.2	7.5	1.5	5.0	1.9	6.30***

TABLE 5.13

16PF Factor A : Tests of differences between means

HSD = 1.3 MSe = 2.55

	Asthmatics	Ref. Ast.	Neurotics
Normals	0.4	1.0	1.0
Asthmatics	-	0.6	0.6
Ref. Ast.		-	0

None of the means differ significantly

TABLE 5.14

16PF Factor B : Tests of differences between means

HSD = 1.5 MSe = 3.84

	Neurotics	Asthmatics	Normals
Ref. Ast.	0.4	1.2	2.1*
Neurotics	-	0.8	1.7*
Asthmatics		-	0.9

TABLE 5.15

16PF Factor C : Tests of differences between means

HSD = 1.6 MSe = 4.37

	Ref. Ast.	Asthmatics	Normals
Neurotics	1.3	2.0*	2.8*
Ref. Ast.	-	0.7	1.5
Asthmatics		-	0.8

TABLE 5.16

16PF Factor L : Tests of differences between means

HSD = 1.8 MSe = 5.01

	Ref. Ast.	Asthmatics	Neurotics
Normals	0.5	0.8	2.3*
Ref. Ast.	-	0.3	1.8*
Asthmatics		-	1.5

TABLE 5.17

16PF Factor O : Tests of differences between means

HSD = 1.7 MSe = 4.48

	Asthmatics	Ref. Ast.	Neurotics
Normals	0.4	1.2	2.8*
Asthmatics	-	0.8	2.4*
Ref. Ast.		-	1.6

TABLE 5.18

16PF Factor Q_4 : Tests of differences between means

HSD = 1.5 MSe = 3.59

	Asthmatics	Ref. Ast.	Neurotics
Normals	0.9	1.1	2.5*
Asthmatics	-	0.2	1.6*
Ref. Ast.		-	1.4

as a trait. For all these scales, the asthmatics are midway between the normals and neurotics; i.e. there is no evidence that any specific personality traits are associated with asthmatics and not with non-asthmatic subjects. However, the two asthmatic groups score lower than the normals and neurotics on factors E, I, M, and N (although the differences between the groups are not significant).

ii. Second order factors

Table 5.19 shows the means scores on the four second order 16 PF factors. The only factor showing any significant differences is I, the Anxiety factor. The tests on the means in table 5.20 show that the neurotics are significantly higher than the other three groups, none of which differ in their mean scores. This result is not unexpected.

2. Comparison of the experimental groups' results with normative data

A comparison of the normal groups' scores with published norms for the psychometric tests demonstrated that they were in some respects "super-normal", showing extremely low scores on the scales measuring anxiety and neuroticism. Since normative data obtained from large groups of subjects was available for all the tests used, all the experimental groups were compared with this normative data, using Student's 't' tests for unmatched groups to test the significance of any differences. The results of these tests are shown in table 5.21, together with details of the published data used.

TABLE 5.19

16PF Questionnaire - Second Order Factors : Group results

Factor	Asthmatics		Referred Asthmatics		Neurotics		Normals		Analysis of variance
	n=67		n=14		n=20		n=22.		
	mean	s.d	mean	s.d	mean	s.d	mean	s.d	F ratio
I	5.9	2.0	6.1	2.7	8.1	2.1	4.8	2.3	8.60***
II	4.8	2.2	4.9	3.0	5.2	2.2	5.1	1.5	0.27
III	5.9	1.5	5.7	2.0	5.4	1.6	6.1	1.5	0.68
IV	7.0	1.5	7.4	1.9	7.5	1.6	7.6	2.1	0.78

TABLE 5.20

16PF Second Order Factor I (Anxiety) : Tests of differences between means

HSD = 1.7, MSe = 4.71

	Asthmatics	Ref. Ast.	Neurotics
Normals	1.1	1.4	3.3*
Asthmatics	-	0.3	2.2*
Ref. Ast.		-	1.9*

Test	Scale	Normative data mean	s.d	t tests Asthmatics	Ref. Ast.	Neurotics	Normals
TMAS (Kelly & Walker, 1968) n=60		13.4	8.2	1.35	2.75**	6.94***	2.25*
EPI (Eysenck & Eysenck, 1964)	N	7.8	4.5	3.87***	2.40*	6.91***	2.72**
	E	11.6	4.9	2.96***	1.69	1.06	2.45*
HDHQ (Philip, 1968) n=240	Total	15.1	6.9	1.17	1.75*	5.95***	2.41*
	Direction	3.1	5.7	0.34	2.20*	1.36	5.65***
16PF (Philip, unpublished data) n=284	A	5.26	1.96	1.40	0.44	0.54	1.77
	B	7.55	1.63	1.09	1.63	1.42	3.21**
	C	5.75	1.86	2.56*	2.63**	6.19***	0.36
	E	5.82	1.18	3.87***	1.20	0.76	1.70
	F	5.80	2.13	0.60	1.19	1.21	0.21
	G	5.11	1.82	0.43	1.02	0.49	0.22
	H	4.95	1.92	0.19	0.28	1.22	1.30
	I	5.68	2.15	3.81***	2.35*	1.78	1.45
	L	5.23	2.02	1.31	0.13	4.02***	0.96
	M	5.74	1.90	0.91	0.27	1.30	0.14
	N	5.42	2.17	2.52*	0.54	0.04	0.68
	O	5.77	1.75	1.12	1.09	5.15***	1.70
	Q_1	5.87	1.85	3.29**	2.44*	1.92	2.74**
	Q_2	5.81	2.14	0.38	0.66	1.23	0.41
	Q_3	5.01	2.08	0.32	0.67	0.44	2.38*
	Q_4	5.38	1.95	1.99*	1.34	4.76***	0.88

It should be noted that the normative data for the 16PF was based on the other full version of the test, form A. Thus these results should be interpreted with caution. It can be seen that the normal group do indeed score lower on the TMAS, EPI Neuroticism Scale and the HDHQ Total Hostility and Direction of Hostility scales. they are also more intelligent (16PF - Factor B), more experimental and radical (Q_1), and controlled (Q_3). In addition, they score lower on the EPI Extraversion scale than the published norms. The results for the neurotic group confirm the differences obtained from the within-study analyses of variance and need no further comment.

The results for the asthmatic group do show some evidence of differences which were not evident from the within-study results. However, interpretation is difficult. They do not score significantly higher on the TMAS, but are more neurotic and more introverted on the EPI, On the 16 PF Questionnaire they are more affected by feelings (Low C), submissive (Low E), tough-minded (Low I), forthright and natural (Low N), more radical (high Q_1), and tense (high Q_4).

Other findings of note are the asthmatics' low scores on factors E and N, and both the asthmatic and referred asthmatic groups' low scores on factor I. Since neither the normal nor the neurotic groups show divergencies on these scales, it can be concluded that these differences are peculiar to the asthmatics. The fact that the referred asthmatic group show <u>fewer</u> departures from the published

norms than the asthmatic group may be due solely to the small numbers in this group. Alternatively, it may be because they are displaying more neurotic traits, which are in conflict (in the sense of displacing the group along a bipolar personality dimension) with the "asthmatic" traits observed. This is certainly true for 16PF factors E and N, where the referral asthmatics are midway between the asthmatic and neurotic groups.

E. Discussion

In contrast to the findings of other workers, the asthmatic group studied here did not show evidence of psychopathology when compared with large groups of normal subjects, with the exception of their above-average score on the EPI Neuroticism scale and 16PF factor Q_4. However, these results obscure the fact that although statistical non-significance was found in many tests, the range of scores obtained by the asthmatics on the scales measuring neuroticism and anxiety was extremely wide. There were several asthmatics who were psychometrically indistinguishable from the neurotic group, and one could surmise that for several of these individuals, their neurotic personality could cause more distress than their pulmonary pathology.

This wide range of scores (which was noted in all groups) clearly reduces the power of single tests in assessing the presence of neurotic psychopathology in both groups of patients and in

individual cases. The latter is particularly important if the question of psychiatric treatment is under consideration. To help overcome the problem, these scores could usefully be combined by a discriminant function analysis, taking the normals and neurotics as criterion groups thus giving one index which would allow a more precise assessment to be made.

There is no obvious explanation for the finding that the normal subjects were more introverted than the general population, except that this may be due to cultural factors. This could also explain why the asthmatics are more introverted, but for this group it could also be said that this was a more general reflection of the more specific divergence found on 16PF factor E (humble and submissive).

The finding that asthmatics are more submissive and humble is in accord with Rees's (1956, op.cit.) observer ratings of personality. Equally interesting is the finding that asthmatics are tough-minded (factor I) rather than tender-minded and dependent which is discordant with the frequent reports of traits of dependency and sensitivity in asthmatic children (though there is no reason to assume that children and adult asthmatics should necessarily show the same traits).

An advantage of the 16PF Questionnaire is that its sixteen source traits can give a more detailed profile than the two dimensions of a test such as the EPI. In this context, it is interesting to note the results of the asthmatics on the traits which comprise the

second order anxiety factor. They differ from the norms on factor C (more affected by feelings) and factor Q_4 (tense and anxious), but do not differ on factors H, L, O and Q_3. Thus there is not much evidence that they suffer from the more psychic components of anxiety (though they are more tense than normal) but at the same time they appear as a group to be more affected by their feelings, when they do occur. This may well be an acknowledgement by many of them that they see emotion as affecting their asthma. The asthmatics' score on 16 PF factor Q_4 is in contradiction to their normal score on the TMAS (which primarily measures the somatic symptoms of anxiety, as does factor Q_4), and their above-average score on the EPI Neuroticism scale is in conflict with their score on 16 PF factor O, which would be expected to contribute to neuroticism.

CHAPTER 6

THE PSYCHOPHYSIOLOGICAL RESPONSE TO A BREATHING STRESS

A. Introduction

In contrast to the two psychophysiological tests described in Chapter 4, this phase of the investigation was not designed to look solely at a measure, or group of measures known to covary mainly with anxiety. It was hoped that through observing the impact of a mild stress it would be possible to examine both the qualitative and quantitative aspects of psychophysiological responsivity in the asthmatics and the control groups. Since the stressor of an airways resistance mildly resembled one aspect of the respiratory distress associated with asthma - being unable to exhale easily - differences in the responses to this stress could be of value in assessing the role of psychological factors in the distress felt during real attacks.

Specifically, it was hoped that this experiment would throw some light on the following questions.

a. Is the magnitude of the asthmatics' responses to a respiratory stress different from that of the non-asthmatics.

b. Are the observed responses consistent enough to justify thinking of response specificity as a characteristic of the individual (or group).

c. Do the patterns of the asthmatics' physiological responses differ from that of the non-asthmatics.

d. Are the qualitative and quantitative aspects of the observed patterns of response related to any psychological characteristics of the individual or group?

B. Method

1. The production of an applied external airway resistance

The airway resistance used as the stressor was produced by an electrically controlled valve (see Figure 6.1). This was connected by rubber tubing to the face mask that the subjects wore. The valve was controlled in the following way. When it was switched on by the experimenters in the laboratory the solenoid was energised and the valve moved from its "normally open" position to one of closure, thus blocking off the airway. A pressure transducer (Greer micro-manometer with a type A300 capsule) was connected to the subject's face-mask. The voltmeter which displayed the mask pressure incorporated a limit sensing device. When the mask pressure became greater than a given value (selected by the experimenter) the limit sensing device operated a relay which was connected to the valve control circuit and caused the valve to stay open for a fixed period, thus permitting expiration to take place. The duration of the opening of the valve was controlled by a delay circuit which could be adjusted to suit different rates of breathing. The control circuit is shown in Figure 6.2, and a record of the mask pressure waveform associated with normal breathing and with breathing against the resistance is displayed in Figure 6.3.

2. Design of the experiment

Each testing session lasted for 21 minutes and was divided into seven three-minute periods. Periods 1,3,5 and 7 were relaxation periods when the subject was breathing freely. Periods 2,4, and 6 were "stress" periods when the subjects were exposed to resistances to expiration corresponding to a pressure of 2,4, or 8 cms. H_2O.

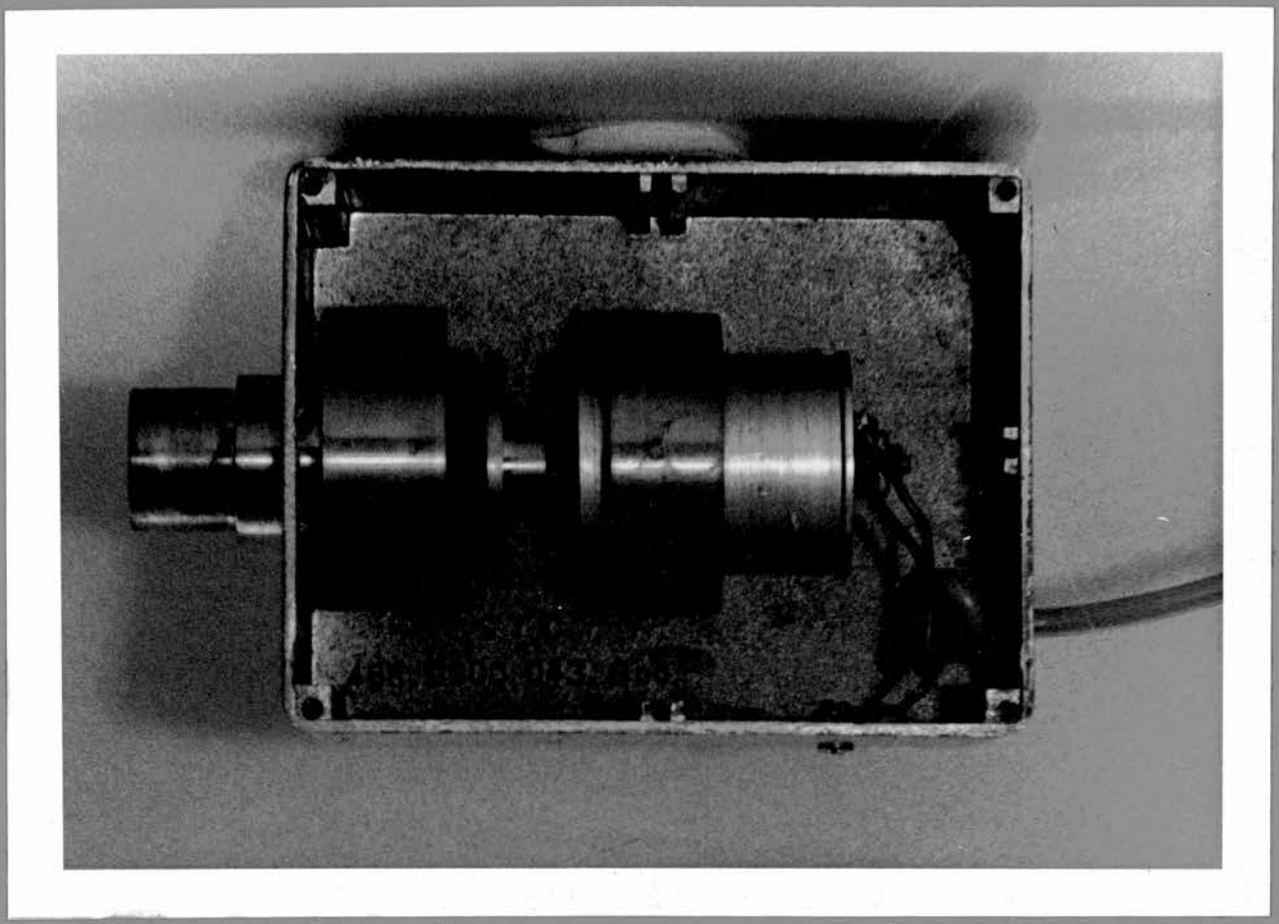

Figure 6.1: The airway resistance valve.

The expiratory tube from the mask is connected to the inlet at the left of the picture. The plunger in the centre blocks expiration when activated by the solenoid on the right.

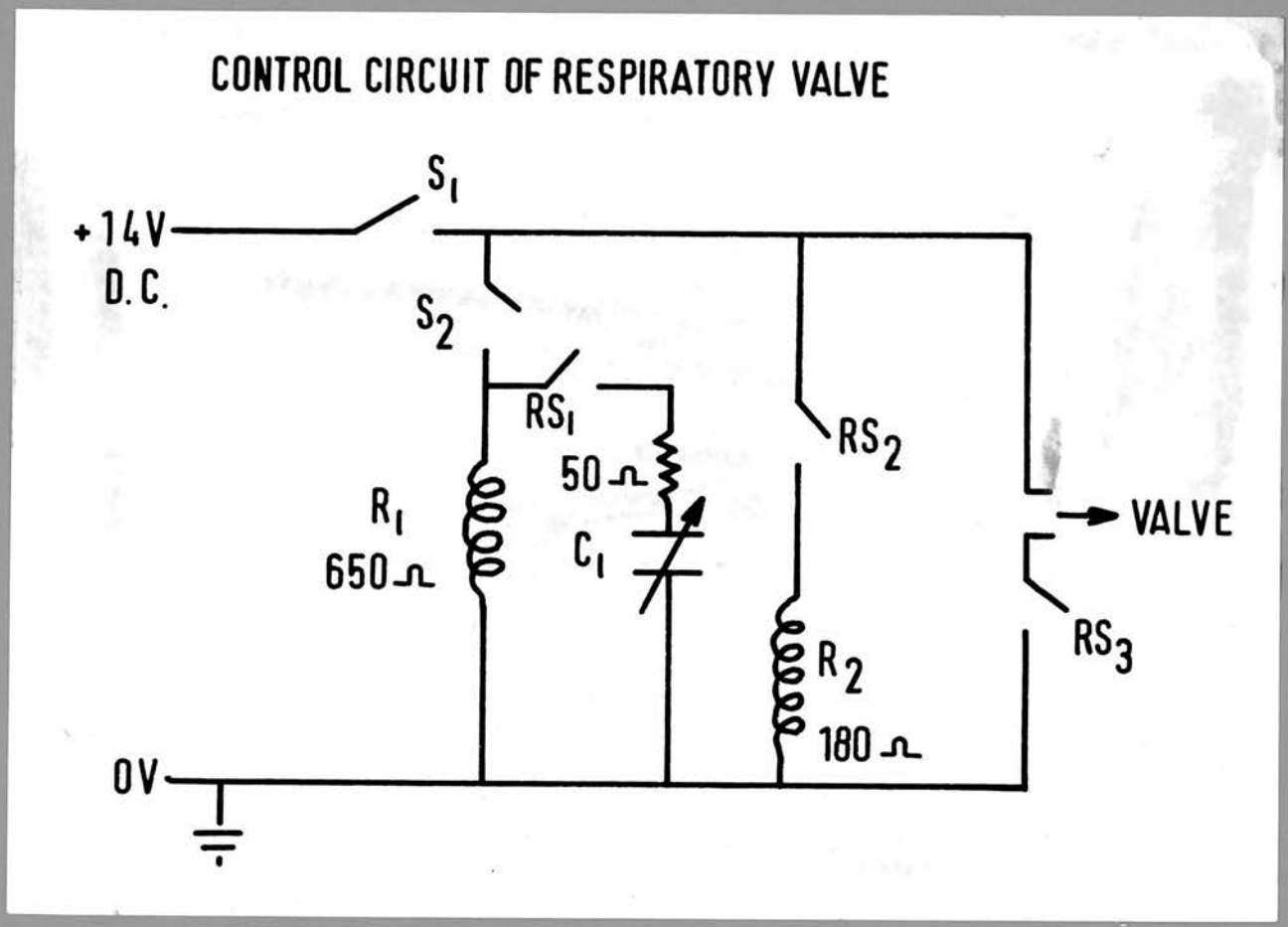

Figure 6.2: Control circuit for valve.

KEY

S_1 - a main valve switch
S_2 - limit switch on voltmeter
R_1 - Relay, 650 , 2 pole 4 way
R2 - Relay, 180 , 2 way

RS_1 RS_2 - relay contacts activated by R_1
RS_3 - relay contact activated by R_2
C_1 - variable capacitance 0-4000 uF

When S_1 is closed, relay contact RS_2 is closed so that R_2 is activated and the valve is closed, through RS_3 being closed. When the subject breathes above the set pressure, S_2 closes and R_1 is activated, opening RS_2 (thus opening the valve) and charging the variable capacitance C_1. When S_2 is opened, C_1 discharges through R_1, keeping it activated. When C_1 has discharged (after a delay controlled by the time constant of R_1C_1 relay R, switches off and RS_2 and RS_3 operate to close the valve.

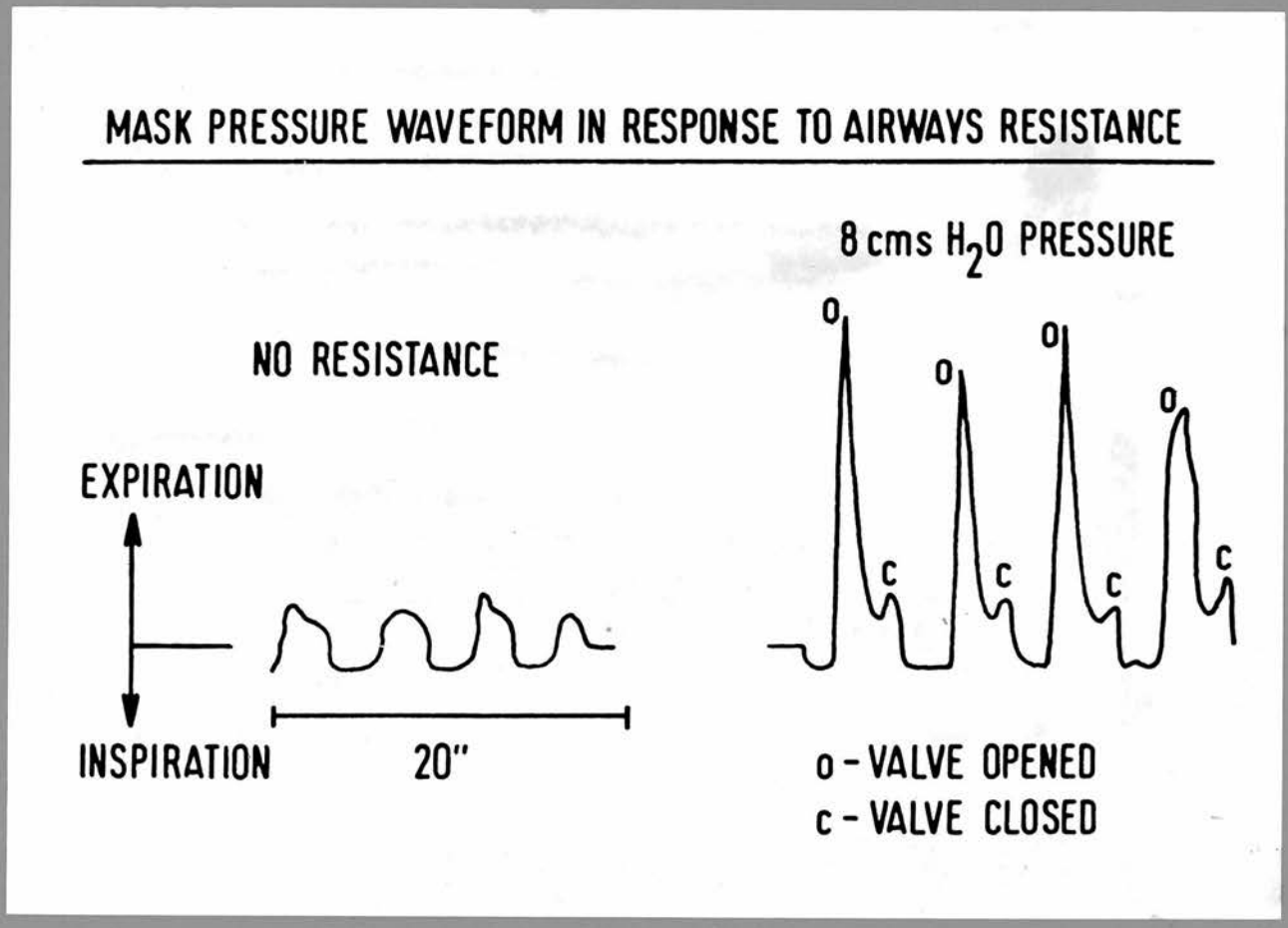

Figure 6.3: Mask pressure wave form.

When the valve is in operation, the mask pressure rises on expiration to the point O, where the valve opens. Pressure then drops while the subject exhales freely, and closes at the point C. The slight increase in pressure at C is probably an artefact caused by the sudden closure of the valve.

It was originally intended that these pressures should be presented in a latin square design so that both order effects and the effects of different stresses could be examined. Because it was impossible to obtain 12 subjects in groups 3 and 4 of the asthmatics, and due to certain difficulties in matching controls to asthmatic subjects this aim was not realised. However, each subject received the stresses in the opposite order on the second occasion of testing. Thus each subject acted as his own control for order effects. The number of subjects receiving each different magnitude of resistance first was the same. Since adaptation of physiological response is such a well demonstrated phenomenon, this deficiency in the design may obscure potentially significant results and place limitations on the conclusions which can be drawn.

3. Physiological measures

Four physiological measures were recorded from the subject. These were skin conductance, heart rate, respiration rate and ventilation volume. The methods for measuring each of these is described below, and the overall laboratory system is shown diagramatically in Figure 6.4.

a. Skin conductance

The method used for measuring skin conductance was described in Chapter 4.

b. Heart rate

An ECG signal was obtained from the subject which was amplified by a Devices AC1 pre-amplifier. From this signal, a beat-to-beat measure of heart rate was obtained from a Devices 2750 ratemeter.

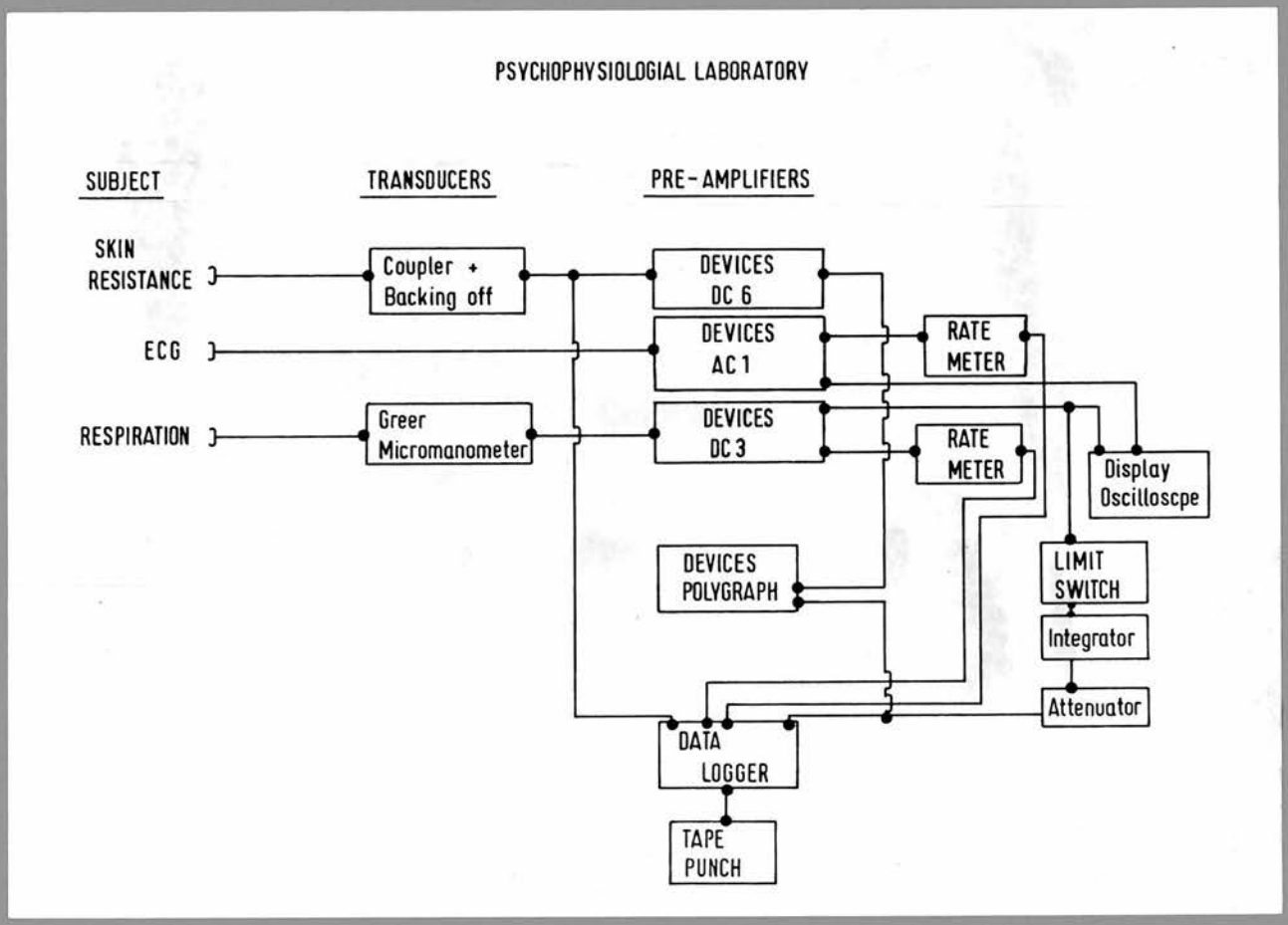

Figure 6.4: Physiological Recording Equipment.

Lead I (left arm - right arm) was normally used; if the signal did not trigger the ratemeter cleanly (usually because the QRS complex was of the same amplitude as the T wave) a leg electrode was attached and another lead (II or III) was used. Since the subjects were earthed through the skin resistance electrodes, no separate earth lead was used.

c. Respiration

The subjects wore a face mask (adapted from R.A.F. type P or Q masks). This uniflow mask had separate valves for inspiration and expiration. A pneumotachograph was attached to the inspiratory inlet. This is a cylinder with a thin piece of gauze mounted inside it which develops a small pressure difference proportional to the flow of air across it into the mask (analogous to the Ohm's law relationship Voltage = current/resistance). This pressure difference was measured by a Greer micromanometer (with a type A10 capsule) whose output is a voltage proportional to the flow. This signal was then amplified by a Devices DC3 pre-amplifier. From this it was possible to measure two respiration parameters. Respiration rate was measured by feeding the flow signal to another Devices ratemeter, giving a reading of breath-to-breath rate. Ventilation volume was measured by passing the flow signal first through a limit switch, which electronically clipped the signal so that it was half wave rectified, then through an electronic integrator, whose output was proportional to the integral of the flow signal over time, and finally through an attenuator which reduced the magnitude of the integrator output to a level suitable for recording on the polygraph.

The integrator was reset either by a timer every 30 seconds or by a level detector which fired when the signal into it (the output from the integrator) reached a preset level. This method of operation was used when the data logger (see below) was operating.

4. Calibration of measuring systems

a. Skin conductance

The calibration circuit in the coupler (see Figure 4.2) was used to check the value of the constant current before every recording.

b. Heart rate and respiration rate

The meters were checked from time to time using an electronic pulse generator, which emitted pulses of a known frequency. Their stability was extremely high.

c. Ventilation volume

The ventilation system was calibrated using a respiratory pump which produced a regular sinusoidal waveform. The pneumotachograph was attached to the pump which was then set in motion. With a known stroke volume and a known rate the size of the integral on the polygraph could be related to a known volume of air. Figure 6.5 shows the linear regression of minute volume against the displacement of the pen produced on the polygraph, together with its 95% confidence limits. The accuracy of the calibration was checked every few months and was found to be stable.

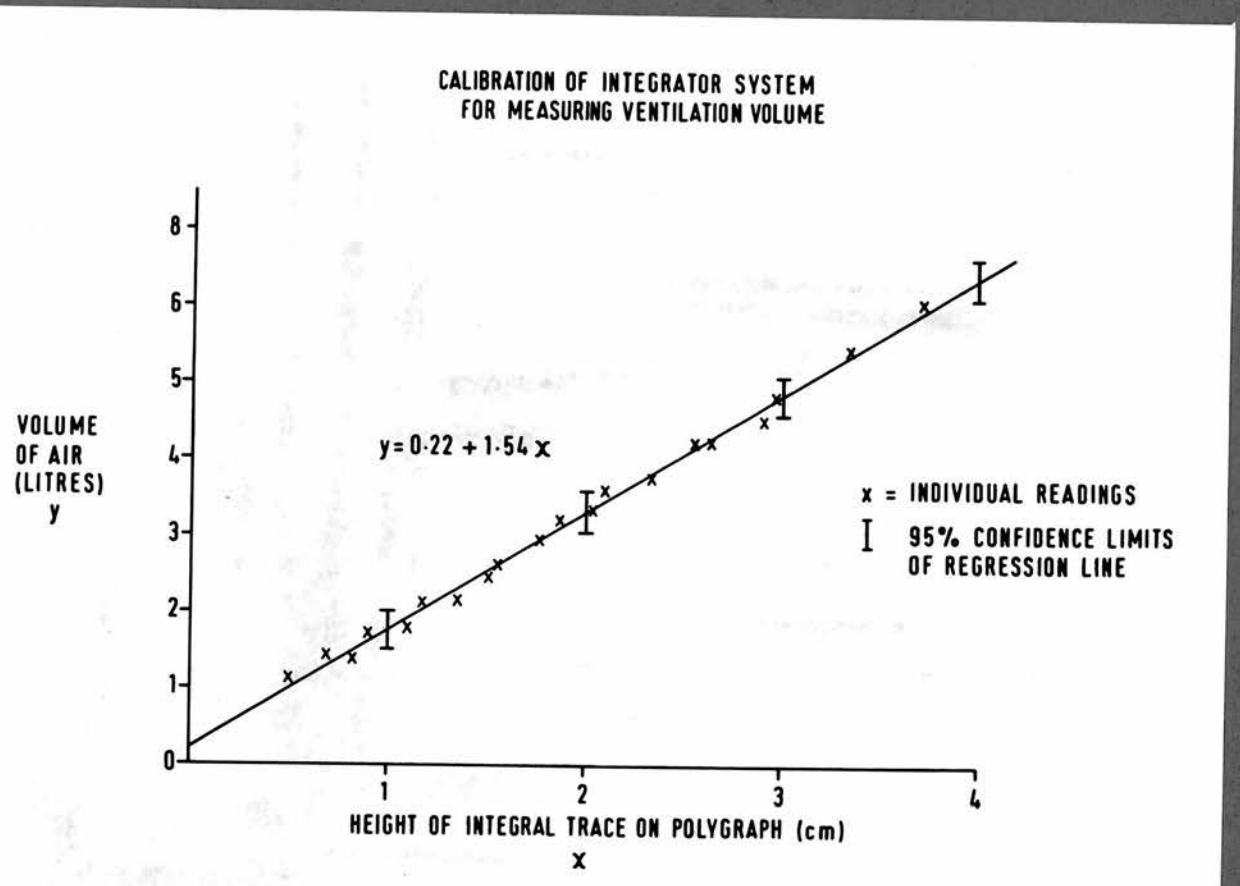

Figure 6.5: Calibration of ventilation volume measuring system.

5. Data logging system

During the study, the data recording facilities available were augmented by the incorporation of a data logging system into the laboratory. Because this is a novel measuring system in the context of psychophysiological research, and because its advent caused some changes in the methods employed for data sampling, it will be described in some detail.

One of the main problems in psychophysiological research is the vast amount of data which can be collected in a very short time. If this is recorded on a polygraph, considerable data reduction, often carried out on a very arbitrary basis, has to be done manually before analysis of data can take place. In the last few years, several systems have been described for direct recording of data onto digital magnetic tape, (Ax, 1962, Zimmer 1966) but these have been expensive both to instal and to operate.

The system chosen for the laboratory was a commercially available instrument, the Solartron "Compact 2" data logger (shown in Figure66). Basically, this consists of a digital voltmeter with a scanner attached, allowing it to sample and to measurd several signals sequentially; a timer which regulates the frequency of scanning, and a paper tape punch which punches the voltages measures in a computer-readable format on computer tape. It is easy to see that this instrument can easily be applied to the task of sampling physiological variables every few seconds. It is not possible to reproduce the waveform of the original signal completely, nor to record phasic responses taking place over a period

Figure 6.6: The data logger

shorter than a few seconds. However, in the context of measuring the changes in level of a physiological measure in response to a stressor lasting a few minutes this is not critical. Lazarus and his colleagues (1962) sampled every 10 seconds in their study of response to a motion picture stress, and the same sampling frequency was adopted here.

Two computer programs were written in the IMP language to analyse the data. One of these programs (READDATA) simply checks the format of each data tape, and stores the validated data on magnetic tape or disc. The second program (called ANALYSIS) retrieves data read by the first program, converts the voltage readings to biological units, divides the data up into periods or epochs and analyses the data in each period or group of periods (producing such statistics as the mean, standard deviation, maximum and minimum value). The progress of the program is controlled by a command language which selects the analyses to be done on each set of data and allows such information as the beginning and end of each period to be given to the program in a form not too far removed from simple English. Two important parameters which the program needs are user-specified maximum and minimum values for each physiological measure based on observations made during the experiment. Any readings falling outside this range are considered as artefacts and are ignored in the analysis. Listings of the two programs can be found in Appendix 2. Checks were made to ensure that manually collected data was comparable with the data obtained from the data logger. These are described in Appendix 3.

6. Data sampling and the choice of response measures

A system capable of gathering and analysing data such as the one described above naturally calls attention to the problem of data sampling and the justification for the practice adopted. It is possible in theory to take a vast number of indices of autonomic activity from a single physiological measurement channel, but the number that can be used in practice is dependent on the time and the facilities available. There are few studies of the independence of measures derived from the same physiological variables. In the one systematic investigation of this problem, Speisman et al. (1961) report an analysis of skin conductance and heart rate measures at rest and while watching a stressful film. The conductance and heart rate measures were analysed in as many ways as was feasible and the resulting scores were subjected to a cluster analysis. The conclusions germane to the present research were that the measures of variability and level were particularly meaningful since they appeared in independent clusters, while such measures as response and recovery time seemed less useful. The various methods used to assess level clustered well together. The authors concluded that "Judicious selection of scoring systems can be tailored to fit given experimental conditions, and there can be considerable confidence that many of the specific techniques are interchangeable."

As mentioned above, the data logger sampled the physiological measures every 10 seconds, but before it was in use, readings of level were taken every 30 seconds. This was the maximum possible rate, both in terms of human information processing capabilities, and the time available to score the polygraph records. The measures derived subsequently from these readings were ones of mean level over a period of a few minutes, so there are no statistical grounds for supposing that the means derived from readings obtained every 30 seconds are more biased estimates than those derived from more frequent readings.

7. Data reduction and analysis

For each physiological variable, nine measures of level were obtained from each 21 minute session. These were

a. The mean of the readings in the 90 seconds preceding each breathing stress.

b. The mean of all readings during each three minute stress period.

c. The mean of all readings during each 90 second post-stress period.

These readings were derived by taking readings every 30 seconds from "hand-logged" data, or by computing the mean of the readings taken every 10 seconds from the data logger records, where these were available.

The measurement of polygraph records, from which skin conductance and ventilation volume data was read, was not checked systematically; however, occasional records selected at random were read by both the

author and the laboratory assistant who was responsible for reading the records. A negligible number of discrepancies were found, and these could invariably be attributed to individual differences in visual acuity rather than to systematic errors.

There were thus nine readings for each physiological variable on each occasion of testing, which was used as the basic data for all the analyses reported in this chapter. Most of the analyses reported in this chapter were carried out using a computer program written in FORTRAN. This calculated the various response measures and computed the various correlations, means, standard deviations etc. A listing of this program is given in Appendix 2.

8. Missing data

Unfortunately, the skin conductance records of 24 subjects (all the asthmatics in group 1 and their controls) could not be used. This was due to the ill-advised use of Cambridge electrode jelly as the contact medium between the electrodes and the skin of these subjects. It was discovered later that this jelly had a very high salt content, and was not isotonic with sweat. Under these circumstances it seemed more than possible that the skin resistance readings obtained would have been inaccurate since Edelburg and Burch (1962) reported that hypertonic electrolytes lead to unusual drifts in skin conductance level over times. It was decided not to attempt to analyse this data.

Other missing data points occurred for the following reasons:

a. the occasional breakdown of equipment before or during a run.

b. the relative insensitivity of the respiration ratemeter to shallow breathing.

c. artefacts in heart rate and skin conductance due to the subject moving.

When equipment breakdown occurred the data was obviously irrevocably lost, except for a period when the respiration ratemeter was out of order, and a "breath-counting" technique was used. This entailed counting the number of breaths per minute for the polygraph record of ventilation volume. Appendix 3 shows that this did not yield significantly different results from ratemeter measurements. "Breath-counting" was also used when b. above occurred.

Since empirical observation showed that the variation in measures of basal level was far greater between than within subjects, it was decided to estimate any missing data points from the rest of the subject's data, provided that not more than one point out of the possible nine per variable was missing. In all, 14 (out of a total of over seven thousand) readings were estimated in this way. The purpose of this was to make feasible the analyses of variance to be reported later.

C. Results

1. A preliminary analysis

The first analysis performed on the data was designed to answer the basic question - "Is there any evidence that the experimental treatment produces a physiological response?". To examine this, an analysis of variance was performed. For each subject, the basal value before and during the stressor of 8 cms. H_2O pressure resistance was taken for each of the four physiological measures separately. The results of the four analyses are shown in Tables 6.1 to 6.4. This particular response was selected on the reasoning that if no response (i.e. difference in basal level) could be shown as a result of the largest stress that occurred during the first testing session (when subjects would not be expected to have shown much adaptation) then there was little point in analysing the data in depth.

The results for all the variables show that the treatment effect is significant. However, only for heart rate is there a significant difference between any of the experimental groups, and none of the Groups x Treatment terms are significant. This is an indication that the full analysis of results would not reveal any response patterns specific to any of the experimental groups.

At this point it was decided not to include the "referred" asthmatic group in any subsequent analyses. This was done because of the small numbers in the group; in addition, it was felt that analysis of this data would contribute little to an understanding of the differences between the asthmatic and non-asthmatic groups.

TABLE 6.1

ANALYSIS OF VARIANCE : SKIN CONDUCTANCE

Source of variance	df	SS'	MS	F
Between subjects	100			
Groups	3	151247	50416	0.62
Subjects in Groups	97	7879105	81228	
Within subjects	101			
Levels	1	16344	16344	26.00***
Groups x Levels	3	1292	431	0.68
Levels x Subjects in Groups	97	60964	629	

TABLE 6.2

ANALYSIS OF VARIANCE : HEART RATE

Source of variance	df	SS	MS	F
Between subjects	124			
Groups	3	4158.88	1386.29	3.57*
Subjects in Groups	121	4699.00	388.35	
Within subjects	125			
Levels	1	334.08	334.08	12.52***
Groups x Levels	3	20.26	6.75	0.39
Levels x Subjects in Groups	121	3227.80	26.68	
	249			

TABLE 6.3

ANALYSIS OF VARIANCE : RESPIRATION RATE

Source of variance	df	SS	MS	F
Between subjects	124			
Groups	3	44.55	14.85	0.36
Subjects in Groups	121	4912.15	40.60	
Within subjects	125			
Levels	1	35.34	35.34	7.95***
Groups x Levels	3	30.04	10.01	2.25
Levels x Subjects in groups	121	537.62	4.44	
	249			

TABLE

TABLE 6.4

ANALYSIS OF VARIANCE : VENTILATION VOLUME

Source of variance	df	SS	MS	F
Between subjects	123			
Groups	3	60.49	20.16	1.43
Subjects in Groups	120	1690.06	14.08	
Within Subjects	124			
Levels	1	407.76	407.76	105.99***
Groups x Levels	3	2.90	0.97	0.25
Levels x Subjects in groups	120	461.66	3.85	
	247			

2. The selection of a response measure

In the analysis of psychophysiological experiments, there are essentially three types of measure which can be obtained to represent a subject's physiological activity. These are

a. measures of basal level

b. measures of response

c. measures of individual non-specific variability.

In analysing the results of this experiment it was decided to concentrate on the first two types of measure, since estimates of variability could possibly have been influenced by the different modes of data collection employed, and would have been difficult to work out for "hand-logged" data.

Basal levels are obviously expressed in the basic measurement units, but the selection of a response measure is not so easy. The main difficulty in measuring a "true" physiological response is the fact that the observed response is often covaried inversely with the pre-stimulus basal level. This "Law of Initial Value" (Wilder, 1958) has prompted much controversial discussion of suitable statistical methods for computing base-free response measures (for a continuing debate, see Lacey (1956), Dykman et al. (1959), Oken and Heath (1963), Block and Bridger (1962), Heath and Oken (1962), Hord et al. (1964), Benjamin (1967). This complex field will not be reviewed here.

It was decided to investigate a variety of response measures before finally choosing one. The measures used were:

Measure	Definition
1	The simple measure of change: R = stress value-pre-stress value.
2	A more complex measure: R = stress value - $\frac{1}{2}$(pre-stress + post-stress value). This attempts to take into account any long-term drift in basal levels.
3	R = log (stress value/pre-stress value)
4	R = 100 (stress value/pre-stress value)
5	The autonomic lability score (ALS). This is fully described by Lacey (1956), and is a base-free measure of reactivity. It is defined by the formula $ALS = 50 + 10(y_z - r_{xy}x_z)\sqrt{(1 - r_{xy}^2)}$ where y_z = standardised measure of level during stress period. x_z = standardised measure of level during pre-stress period. r_{xy} = correlation between pre-stress and stress levels.

The values for each group were standardised on the basis of the calculated group mean and standard deviation. Lacey does not make it explicit from what combination of readings the ALS would be derived in a situation where the reactivity of groups is to be compared. Clearly, each individual's ALS score should not be computed on the sole basis of the data for the group he belonged to; since the mean ALS for each group

would be 50. It seemed equally ill-advised to work out the scores on the basis of data standardised across all groups, since any systematic group differences in both level and the degree of dependence of final level on initial would be obscured. One possible solution would be to use the normal group's data as the basis for the computation of ALS's of all three groups. This would allow estimates of both individual and group over- and under-reactivity, with direct comparisons with the normal subjects. However, for this particular investigation, each group was to be considered separately, and only the correlations of the different response measures with each other were of interest. Since it was technically easier to compute each individual's ALS from the standardised data of the group alone to which he or she belonged, this procedure was adopted. It also ensured that a series of response measures guaranteed to show zero correlation with basal levels was available as a useful yardstick by which to judge the other response measures.

Each of the five responses was computed for each subject for the three stresses of breathing against 2,4, or 8 cm. water pressure in the first session, and the following measures were computed:

a. the correlation of each response with the four other responses calculated for that stress

b. its correlation with the pre-stress basal level.

The results for each measure are shown in Tables 6.5 to 6.16.

PRODUCT MOMENT CORRELATIONS BETWEEN DIFFERENT RESPONSE MEASURES: SKIN CONDUCTANCE

Table 6.5
Asthmatics n=56

measure

	1	2	3	4	5
1	-				
	89				
2	72	-			
	61				
	98	88			
3	97	70	-		
	98	53			
	99	89	100		
4	98	71	100	-	
	97	51	100		
	99	90	98	99	
5	100	71	97	97	-
	99	59	98	98	
	-12	07	-6	-7	00
basal	-04	-09	-3	-4	00
	-14	04	-22	-22	00

Correlations greater than 0.26 are significant at $p \leq 0.05$.

Table 6.6
Neurotics n=16

Measure

	1	2	3	4	5
1	-				
	90				
2	83	-			
	86				
	97	85			
3	99	80	-		
	95	74			
	98	87	100		
4	99	80	100	-	
	96	75	100		
	99	88	98	99	
5	93	90	91	91	-
	99	81	97	97	
	-13	25	00	02	00
basal	-36	4	-38	-38	00
	14	43	-06	-06	00

Correlations greater than 0.50 are significant at $p \leq 0.05$.

Table 6.7
Normals n=16

measure

	1	2	3	4	5
1	-				
	92				
2	89	-			
	93				
	100	92			
3	100	85	-		
	100	93			
	100	93	100		
4	100	85	100	-	
	100	93	100		
	100	92	100	100	
5	100	89	099	099	-
	100	92	099	099	
	01	02	06	-05	00
basal	-06	06	-07	-08	00
	-09	11	02	-12	00

Correlations greater than 0.50 are significant at $p \leq 0.05$.

All correlations are multiplied by 100.

PRODUCT MOMENT CORRELATIONS BETWEEN DIFFERENT RESPONSE MEASURES: HEART RATE

Table 6.8
Asthmatics n=68

	measure 1	2	3	4	5
1	-				
2	80 80 81	-			
3	99 99 99	81 79 78	-		
4	99 99 99	81 79 77	100 100 100	-	
5	98 100 100	84 81 82	98 99 98	98 99 97	-
basal	-21 -05 -10	06 -07 11	-15 -06 -19	-15 -06 -18	00 00 00

Correlations greater than 0.24 are significant at $p \leqslant 0.05$.

Table 6.9
Neurotics n=22

	measure 1	2	3	4	5
1	-				
2	84 94 92	-			
3	98 99 98	80 94 88	-		
4	97 99 98	79 94 89	100 100 100	-	
5	100 98 100	84 94 91	98 96 98	98 96 98	-
basal	03 -20 05	18 -13 10	-8 -24 -07	-9 -37 -06	00 00 00

Correlations greater than 0.42 are significant at $p \leqslant 0.05$.

Table 6.10
Normals n=22

	measure 1	2	3	4	5
1	-				
2	91 90 65	-			
3	99 100 98	89 88 56	-		
4	100 100 98	89 88 56	100 100 100	-	
5	100 100 98	93 91 56	99 99 99	99 100 99	-
basal	-09 -04 18	15 15 59	-10 -05 +04	-09 -04 09	00 00 00

Correlations greater than 0.42 are significant at $p \leqslant 0.05$.

All correlations are multiplied by 100.

PRODUCT MOMENT CORRELATIONS BETWEEN DIFFERENT RESPONSE MEASURES: RESPIRATION RATE

Table 6.11

Asthmatics n=68

	measure 1	2	3	4	5
1	-				
2	91 68 85	-			
3	95 92 93	85 64 79	-		
4	92 93 96	80 67 81	98 98 98	-	
5	96 98 98	87 65 84	93 91 91	89 92 93	-
basal	-29 -19 -19	-26 -21 -15	-22 -13 -20	-24 -15 -23	00 00 00

Correlations greater than 0.24 are significant at $p \leqslant 0.05$.

Table 6.12

Neurotics n=21

	measure 1	2	3	4	5
1	-				
2	95 94 83	-			
3	98 93 94	93 86 78	-		
4	93 85 85	89 78 70	98 98 97	-	
5	75 49 64	64 41 72	75 50 70	96 45 62	-
basal	-67 -87 -77	-71 -85 -48	-63 -78 -64	-55 -72 -59	00 00 00

Correlations greather than 0.43 are significant at $p \leqslant 0.05$.

Table 6.13

Normals n=22

	measure 1	2	3	4	5
1	-				
2	90 86 81	-			
3	96 97 97	85 81 78	-		
4	95 95 95	82 78 75	100 99 99	-	
5	94 99 98	81 86 77	91 94 98	90 92 96	-
basal	-52 -10 -49	-55 -06 -46	-48 -21 -37	-46 -23 -34	00 00 00

Correlations greater than 0.42 are significant at $p \leqslant 0.05$.

All correlations are multiplied by 100.

PRODUCT MOMENT CORRELATIONS BETWEEN DIFFERENT RESPONSE MEASURES: VENTILATION VOLUME

Table 6.14
Asthmatics n=68

	measure 1	2	3	4	5
1	-				
	78				
2	84	-			
	93				
	91	69			
3	90	64	-		
	94	87			
	85	64	98		
4	88	61	99	-	
	92	83	95		
	100	79	90	84	
5	99	81	93	91	-
	100	93	93	90	
	-04	26	-24	-28	00
basal	-11	38	-22	-23	00
	-01	-03	-28	-25	00

Correlations greater than 0.24 are significant at $p \leqslant 0.05$.

Table 6.15
Neurotics n=22

	measure 1	2	3	4	5
1	-				
	87				
2	88	-			
	98				
	95	84			
3	94	80	-		
	94	94			
	94	86	99		
4	87	73	96	-	
	89	89	95		
	100	87	96	95	
5	85	87	74	71	-
	98	97	87	82	
	07	-04	-04	-03	00
basal	-52	-25	-60	-51	00
	-21	-14	-41	-41	00

Correlations greater than 0.42 are significant at $p \leqslant 0.05$.

Table 6.16
Normals n=22

	measure 1	2	3	4	5
1	-				
	96				
2	95	-			
	94				
	92	85			
3	93	85	-		
	90	76			
	93	87	99		
4	92	83	99	-	
	87	72	99		
	97	91	97	97	
5	97	87	96	95	-
	99	92	93	90	
	26	32	-07	-04	00
basal	26	41	-02	-02	00
	10	26	-30	-34	00

Correlations greater than 0.42 are significant at $p \leqslant 0.05$.

All correlations are multiplied by 100.

It can be seen from all the correlation matrices (except that of the neurotics' respiration rate responses) that the correlations between different response measures are very high. This implies that all these methods of computation are essentially equivalent.

An analysis of the correlations between responses and pre-stress levels shows that, on the whole, responses in this study are independent of basal levels. Exceptions to this occur with respect to the respiration rate responses for normal and neurotic subjects, the normals' responses to stresses of 2 cms. and 8 cms. water pressure, and all the neurotics' responses, all of which show significant dependence on pre-stress level. It is notable that a logarithmic or a percentage transformation of the data does not reduce this dependency.

In spite of this demonstration of the "Law of Initial Values", it was decided to adopt the simple measure - response = stress level - pre-stress level, in all cases. This was chosen, rather than the autonomic lability score, because of its ease in calculation, and because of the difficulties mentioned above in the choice of the appropriate ALS.

3. Comparison of basal levels

To examine differences in basal levels between groups, a one-way analysis of variance was performed for each physiological variable. The measure of basal level used was the "pre-8 cm. stress" period. The results of the analyses are shown in tables 6.17 to 6.20. It can be seen that only for basal heart rate do the groupsdiffer

TABLE 6.17

Analysis of variance of skin conductance levels

(units : Log micromhos)

	mean	s.d	n
Asthmatics	0.825	0.220	56
Neurotics	0.756	0.220	16
Normals	0.841	0.074	16

Source of variance	df	SS	MS	F
Between groups	2	72693	36347.5	0.89 (N.S)
Within groups	85	3460377.8	40710.3	
	87			

TABLE 6.18

Analysis of variance of heart rate levels

(units : beats/minute)

	mean	s.d	n
Asthmatics	78.8	13.2	68
Neurotics	88.5	18.0	22
Normals	75.5	10.5	22

Source of variance	df	SS	MS	F
Between groups	2	2164.96	721.65	4.22**
Within groups	109	18654.4	171.4	
	111			

TABLE 6.19

Analysis of variance of respiration rate

(units : breaths/minute)

	mean	s.d	n
Asthmatics	14.1	4.9	68
Neurotics	15.7	5.5	22
Normals	13.9	4.4	22

Source of variance	df	SS	MS	F
Between groups	2	54.56	27.28	1.12 (N.S)
Within groups	109	2658.76	24.39	
	111			

TABLE 6.20

Analysis of variance of ventilation volume levels

(units : litres/minute)

	mean	s.d	n
Asthmatics	7.0	2.2	67
Neurotics	7.6	3.6	22
Normals	6.3	1.8	22

Source of variance	df	SS	MS	F
Between Groups	2	19.74	9.87	1.58 (N.S)
Within Groups	108	674.21	6.24	
	110			

significantly. The neurotic subjects have significantly higher heart rates than the normal group. This is in line with the findings of other workers (Lader and Wing, 1966), and clinical observations of anxious patients.

4. Analysis of responses

The mean response and its standard deviation to each of the stresses for each group is shown in table 6.21 to 6.24, together with the value of the Student's 't' statistic testing the hypothesis that the response differs from zero. It can be seen that the response to the stress of 8 cm. water pressure in all measures is significant. Other notable features of the responses are:

a. breathing against a stress of 8 cms. water pressure is associated with a decrease in respiratory rate (except for normal subjects in session 2). It appears that this is an adjustment of breathing pattern allowing more "efficient" breathing under the stress, with fewer, but larger, breaths.

b. The neurotics show fewer (8 out of 24) significant responses than the other groups (normals: 11 out of 24, asthmatics: 15 out of 24). This is in line with observations that neurotics are less reactive to stress.

5. Comparison of groups and responses to different stressors

To ascertain whether there were any differences between groups and between different stressors, a further analysis of variance was performed on the response data for the first session. This was a similar design

TABLE 6.21

SKIN CONDUCTANCE: RESPONSES TO STRESS

	Asthmatics n=56			Neurotics n=16			Normals n=16		
Session 1	mean	s.d	t	mean	s.d	t	mean	s.d	t
2 cm. H_2O	-37	271	1.02	51	503	0.41	-16	270	0.63
4 cm. H_2O	-12	235	0.38	-64	180	1.43	135	326	1.66
8 cm. H_2O	185	350	3.96***	214	376	2.28*	229	351	2.61*
Session 2		n=32			n=16			n=16	
2 cm. H_2O	-111	255	2.46*	89	453	0.78	-19	162	0.46
4 cm. H_2O	-76	245	1.75	46	207	0.90	24	401	0.24
8 cm. H_2O	170	397	2.42*	189	640	1.18	104	197	2.11*

units = log micromhos x 10^4

TABLE 6.22

HEART RATE: RESPONSES TO STRESS

	Asthmatics n=68			Neurotics n=22			Normals n=22		
	mean	s.d	t	mean	s.d	t	mean	s.d	t
Session 1									
2 cm. H_2O	-0.3	4.3	0.67	1.3	3.7	1.90	0.6	4.5	0.66
4 cm. H_2O	0.7	4.3	1.28	0.6	5.3	0.53	1.0	4.1	1.09
8 cm. H_2O	2.4	4.8	4.16***	2.3	5.1	2.14*	3.0	2.6	5.24***
Session 2		n=44			n=22			n=22	
2 cm. H_2O	-0.5	3.7	0.82	1.2	4.8	1.21	-0.3	2.7	0.55
4 cm. H_2O	1.3	2.9	2.86**	1.8	4.5	1.91	1.1	2.0	2.69*
8 cm. H_2O	1.8	4.0	2.91**	3.5	5.5	3.00**	2.6	4.1	2.98**

units = beats/minute.

TABLE 6.23

RESPIRATION RATE: RESPONSES TO STRESS

	Asthmatics			Neurotics			Normals		
		n=68			n=21			n=22	
	mean	s.d	t	mean	s.d	t	mean	s.d	t
Session 1									
2 cm. H_2O	-0.7	3.0	1.93	-0.6	4.7	0.69	0.3	2.5	0.50
4 cm. H_2O	-0.2	3.0	0.44	-2.1	5.6	1.63	0.3	2.0	0.75
8 cm. H_2O	-0.2	3.1	0.43	-2.0	3.3	2.77*	-1.0	2.4	1.95
Session 2		n=43			n=22			n=22	
2 cm. H_2O	1.0	2.8	2.40*	0.9	2.6	1.62	0.2	2.3	0.47
4 cm. H_2O	0.5	3.1	0.99	-0.1	2.5	0.26	0.4	3.1	0.61
8 cm. H_2O	-0.5	3.0	1.01	-1.1	3.7	1.38	0.3	2.5	0.59

units = breaths/minute.

TABLE 6.24

VENTILATION VOLUME: RESPONSES TO STRESS

	Asthmatics n=68			Neurotics n=22			Normals n=22		
	mean	s.d	t	mean	s.d	t	mean	s.d	t
Session 1									
2 cm. H_2O	1.2	1.6	6.21***	0.2	1.7	0.65	1.3	1.9	3.20**
4 cm. H_2O	1.6	1.8	7.12***	1.1	2.0	2.40*	1.1	1.9	2.69*
8 cm. H_2O	2.7	2.8	8.13***	2.6	3.6	3.38**	2.2	2.0	5.40***
Session 2		n=44			n=22			n=22	
2 cm. H_2O	1.2	1.7	4.60***	1.0	2.5	1.79	0.9	1.3	3.29**
4 cm. H_2O	1.1	1.6	4.67***	1.7	3.3	2.37*	0.5	0.9	2.47*
8 cm. H_2O	1.8	2.2	5.42***	2.0	2.9	3.22**	1.8	1.6	5.31***

units = litres/minute.

to the one reported in section 6.1, with repeated measures (3 different stressors) within subjects, and the subjects nested within groups. Since there are unequal numbers in the groups, which are not due to any effects of the experimental treatment, the method of unweighted means was used in the analysis, as recommended by Winer (1962, p.374).

The results are shown in Tables 6.25 to 6.28. It can be seen that while all the treatments effects are significant, only respiration rate responses differ between groups. This is due to the neurotics responding with a larger decrease in respiration than the other two groups. Thus one can conclude that being asthmatic does not ipso facto cause any differences in response magnitude to a breathing stress.

6. The consistency of response and basal level measurements

If physiological measures do have any consistent relationship with personality traits, then it would be expected that they should show some individual consistency, both within sessions, and between them. To test this, the following measures were computed:

a. Basal levels:

i) For each variable, the Kendall coefficient of concordance W and the associated value of chi-square (Siegel, 1956) was computed between the nine measurements of level obtained in the first session, taking each of the three groups separately. The results are given in table 6.29.

TABLE 6.25

Analysis of variance of skin conductance responses

Source of variance	df	SS	MS	F
Between subjects	87			
Groups	2	1935	967.5	0.85
Subjects in Groups	85	98113	1154.2	
Within subjects	176			
Stresses	2	22842	11421.0	13.3***
Stresses x Groups	4	3047	761.8	0.89
Stresses x Subjects in Groups	170	145722	857.2	
	263			

TABLE 6.26

Analysis of variance of heart rate responses

Source of variance	df	SS	MS	F
Between subjects	111			
Groups	2	26.55	13.27	0.61
Subjects in Groups	159	2384.85	21.88	
Within subjects	224			
Stresses	2	244.25	122.13	6.88**
Stresses x Groups	4	24.79	6.20	0.35
Stresses x Subjects in Groups	218	3686.08	17.74	
Total	335			

TABLE 6.27

Analysis of variance of respiration rate responses

Source of variance	df	SS	MS	F
Between subjects	110			
Groups	2	157.2	78,6	4.12*
Subjects in Groups	108	2060.4	19.1	
Within subjects	222			
Stresses	2	62.0	31.0	5.03**
Stresses x Groups	4	14.1	3.5	0.57
Stresses x Subjects in Groups	216	1332.9	6.2	
Total	332			

TABLE 6.28

Analysis of variance of ventilation volume responses

Source of variance	df	SS	MS	F
Between subjects	111			
Groups	2	1266.4	633.2	0.70
Subjects in Groups	109	98070.0	899.7	
Within subjects	224			
Stresses	2	12328.6	6164.3	23.09***
Stresses x Groups	4	1468.1	367.0	1.38
Stresses x Subjects in Groups	218	58189	266.9	
Total	335			

TABLE 6.29

Co-efficients of concordance
between within session measurements of basal level

	Asthmatics		Neurotics		Normals	
	W	X^2	W	X^2	W	X^2
Skin Conductance	0.97	481 n=56	0.91	122 n=16	0.87	117 n=16
Heart Rate	0.92	553 n=68	0.96	182 n=22	0.91	173 n=22
Respiration Rate	0.85	568 n=68	0.73	137 n=22	0.84	159 n=22
Ventilation Volume	0.73	438 n=68	0.78	148 n=22	0.68	128 n=22

All values of X^2 are significant at p = 0.001

ii) Product-moment correlations between the "pre-8" basal level of sessions 1 and 2 were computed for each of the three groups for each physiological variable. Results are given in Table 6.30.

TABLE 6.30

BETWEEN SESSION CORRELATIONS OF MEASURES OF BASAL LEVEL

	Asthmatics	Neurotics	Normals
Skin conductance	-0.05	0.41	0.38
Heart rate	0.69***	0.85***	0.55**
Respiration rate	0.79***	0.59***	0.83***
Ventilation volume	0.37*	0.72***	0.65**

b. Responses:

i) Product moment correlations were calculated between the three combinations of response measures taken in pairs in the first session, for each of the three groups. The results are shown in Table 6.31.

ii) Product moment correlations between the response to the 8 cm. H_2O stressor in sessions 1 and 2 were computed for each of the physiological variables, and are displayed in Table 6.32.

TABLE 6.31

PRODUCT MOMENT CORRELATIONS OF RESPONSES TO DIFFERENT STRESSORS WITHIN SESSION I

	Asthmatics	Neurotics	Normals
Skin Conductance			
	n = 56	n = 16	n = 16
2 cm./4 cm.	24	-65**	08
2 cm./8 cm.	08	-02	49*
4 cm./8 cm.	17	-29	37
Heart Rate			
	n = 68	n = 22	n = 22
2 cm./4 cm.	-01	-41	23
2 cm./8 cm.	-02	22	23
4 cm./8 cm.	29*	00	27
Respiration Rate			
	n = 68	n = 21	n = 22
2 cm./4 cm.	36**	71***	33
2 cm./8 cm.	26*	21	71***
4 cm./8 cm.	45***	36	16
Ventilation Volume			
	n = 68	n = 22	n = 22
2 cm./4 cm.	43***	34	85***
2 cm./8 cm.	40***	16	60***
4 cm./8 cm.	50***	68***	66***

* = $p \leq 0.05$
** = $p \leq 0.01$
*** = $p \leq 0.001$

All correlations are multiplied by 100

TABLE 6.32

BETWEEN SESSION CORRELATIONS OF MEASURES OF RESPONSE

	Asthmatics	Neurotics	Normals
Skin conductance	-0.30	0.43	-0.25
Heart Rate	0.06	0.56*	0.09
Respiration rate	0.55	0.44	0.31
Ventilation volume	0.54	0.70	0.57

It is apparent that measures of basal level are highly reliable over short periods of 20 minutes. All of the X^2 values associated with the coefficients of concordance are significant at the $p = 0.001$ level. This implies that any one of the 9 measures of basal level can be selected as a reliable estimate of that parameter for an individual.

The correlations between the two sessions are also high, with the exception of the skin conductance ones. There is no apparent explanation for this.

The reliability of responses within sessions is much less satisfactory. One possible explanation for this is that the readings which are being correlated, although operationally defined as "responses", may in fact be equivalent to a randomly varying quantity. The reason for this is that statistically significant responses to each stressor were not variably obtained (see Tables 6.21 - 6.24); thus, the "responses" are equivalent to the differences between any two readings of level, which would be expected to be randomly distributed around zero. It is notable that the respiratory variables show higher reliability than heart rate and skin conductance.

The correlations between the two sessions are equally disappointing. Only the neurotic group show a significant correlation in heart rate response and none of the group show a significant correlation in skin conductance response between sessions. The normal group's respiration rate response correlation is not significant, otherwise all respiratory responses correlate significantly across sessions.

Thus it cannot be said that there is any evidence of consistency of skin conductance basal level or response, nor of heart rate response. It is difficult to think of reasons to explain this finding. One is that the measure of response is a crude one, since it is based on the average of readings taken over 3 minutes. Since the stressor used directly affected breathing, it is reasonable to suppose that the changes in respiration would last for the whole time that the stressor was applied; thus a mean estimate of respiration rate or ventilation volume over 3 minutes would be quite an appropriate measure on which to base the measure of response. However, there is no a priori reason for assuming that heart rate and skin conductance should necessarily behave similarly. It is possible that, at least in some individuals, they may peak and then gradually decline over the stressor period; thus a measure of the maximum heart rate or skin conductance observed minus the pre-stress level could be a much more sensitive measure of response.

7. Physiological patterning

To examine response and basal level patterns either in individuals or groups, the units of the different physiological variables must be made comparable by some form of standardisation procedure. To do this, a method adapted from one used previously by Engel (1960) was employed. This involved using mean and error terms derived from the preceding analyses of variance. For the assessment of basal level patterns, the mean level for all subjects and the within group variance were taken as estimates of the mean and variance of all subjects' basal levels. For the assessment of response patterns, the mean response (computed from the weighted means data) and the Response x Subjects within Groups mean square error term were taken as the estimates. The actual figures used are shown in Table 6.33.

TABLE 6.33

RESPONSE AND BASAL LEVEL VALUES USED TO STANDARDISE DATA

Variable	Basal Level		Response		
	mean	s.d.	mean	s.d.	
Skin conductance	0.815	0.202	0.0071	0.0294	(log microhmo)
Heart rate	80.0	13.1	+1.33	4.21	(beats/minute)
Respiration rate	14.4	4.9	-0.46	2.48	(breaths/minute)
Ventilation volume	7.0	2.5	1.55	1.63	(litres/minute)

The basal level measures and the responses of each subject in both sessions 1 and 2 were converted to standard scores, with a mean of 50 and a standard deviation of 10.

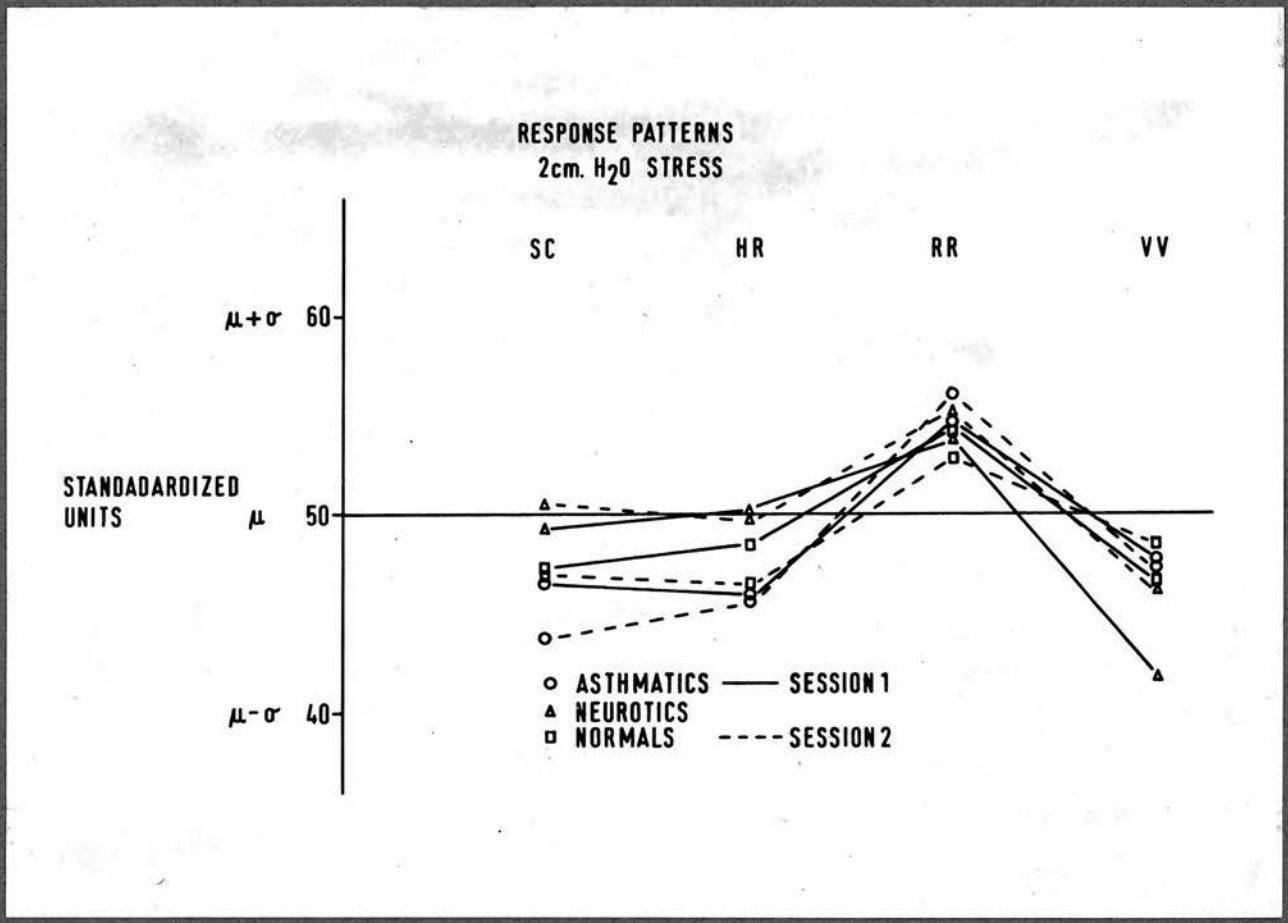

Figure 6.7: Response patterns to 2 cm. H_2O stressor.

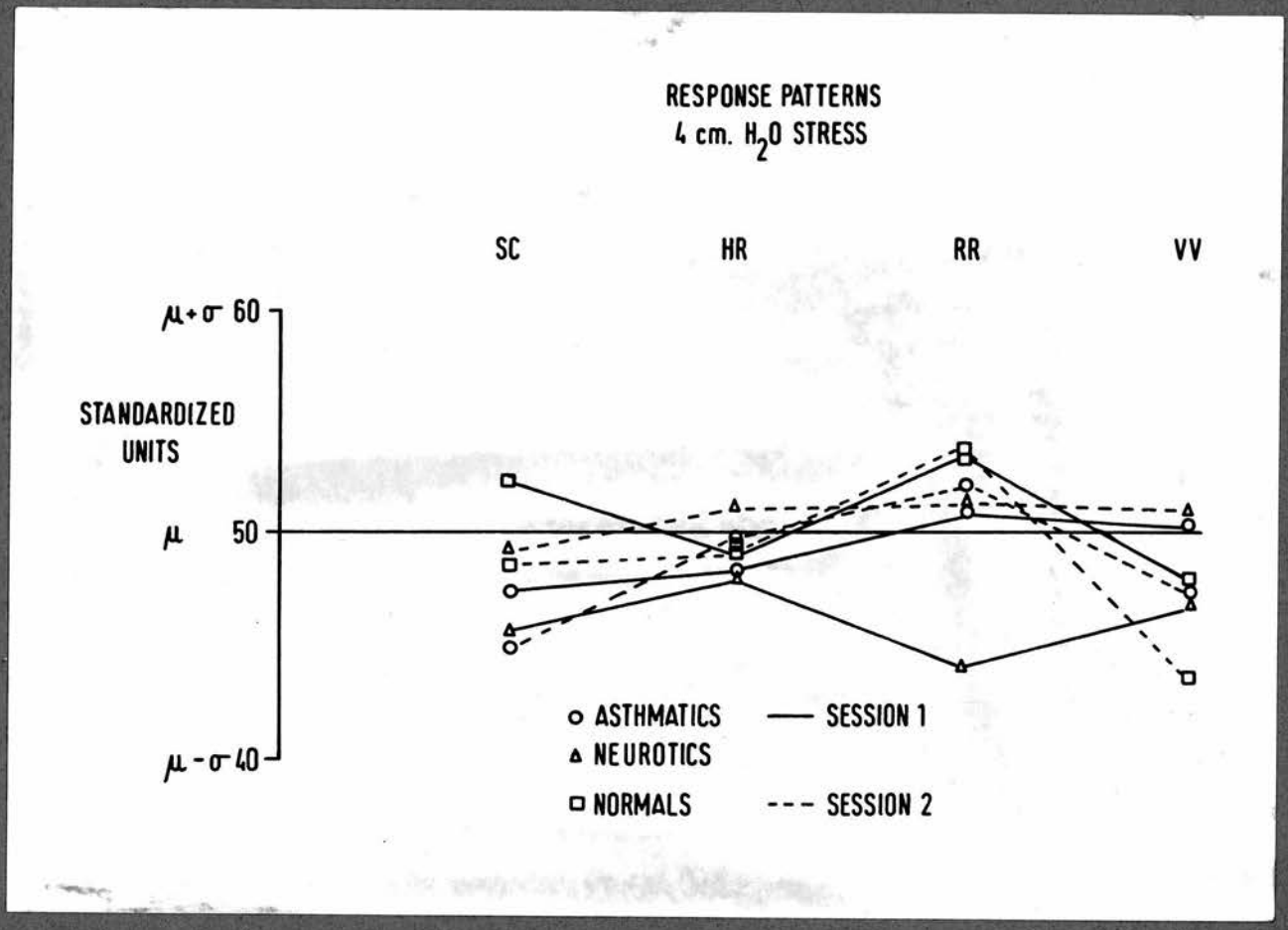

Figure 6.8: Response patterns to 4 cm. H_2O stressor.

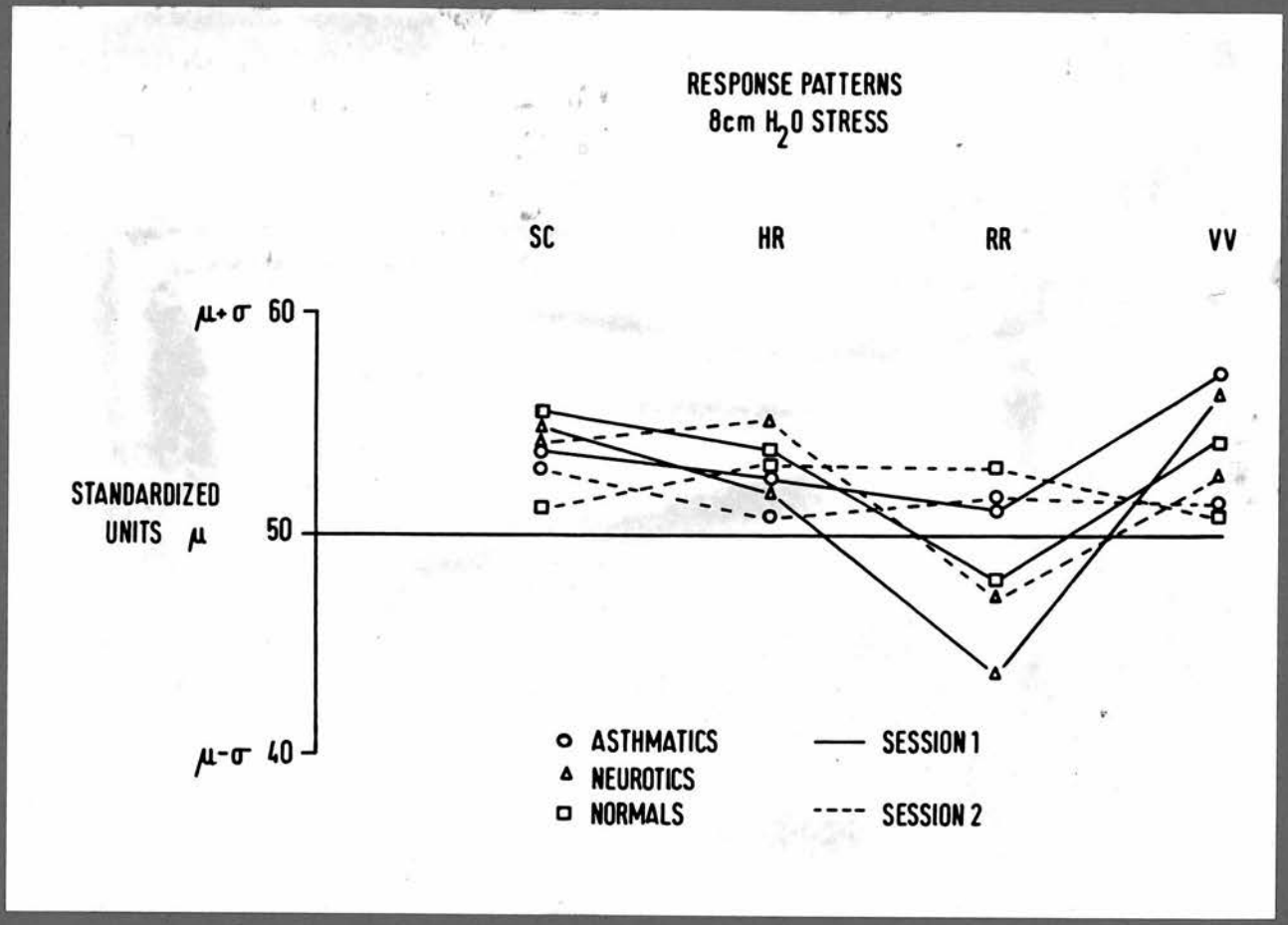

Figure 6.9: Response to 8 cm. H_2O stressor.

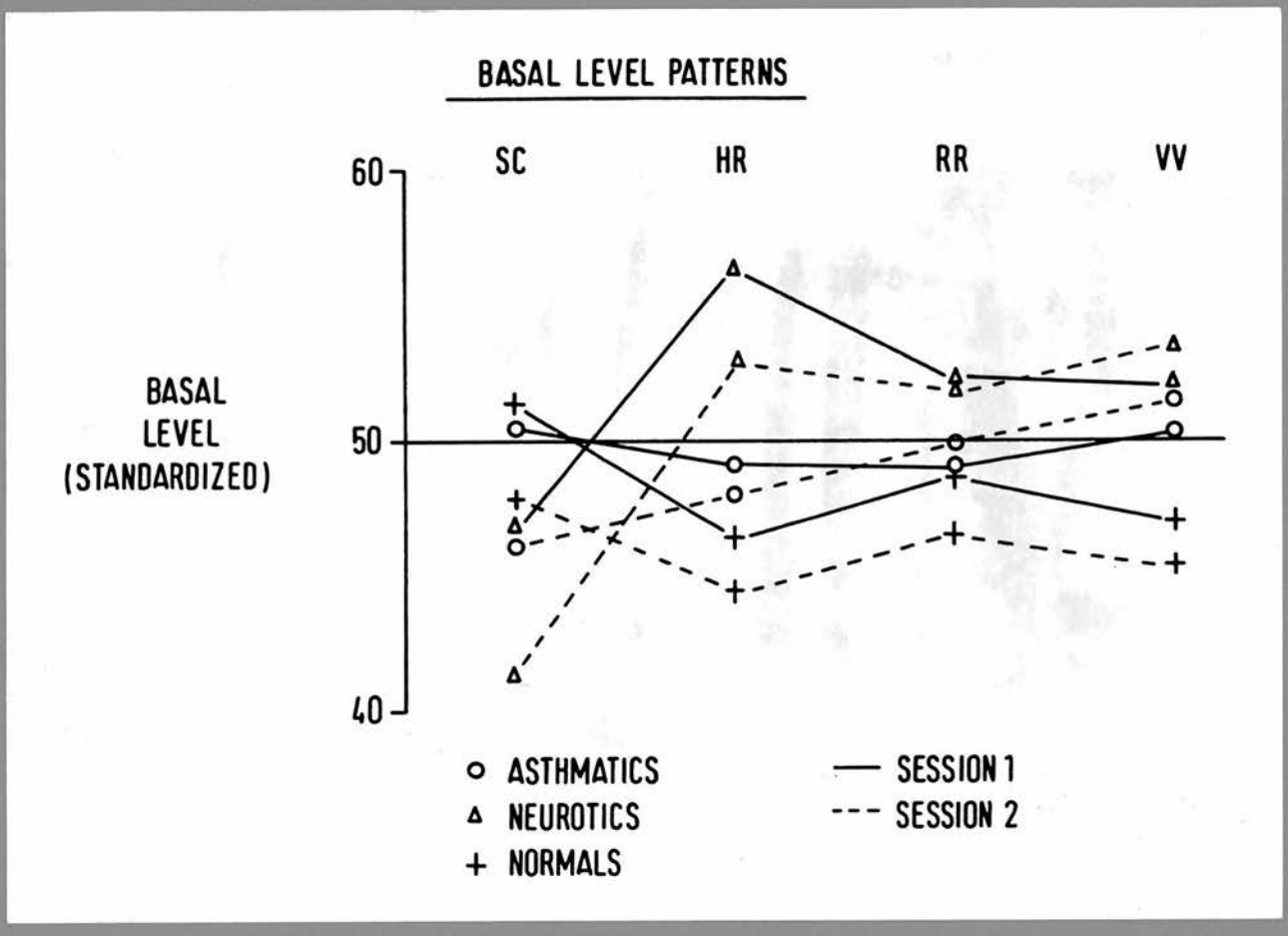

Figure 6.10: Basal level patterns.

a. Response patterning:

The pattern of responses for the three groups for each of the three stresses is shown graphically in Figures 6.7 to 6.9, and the numerical values are given in Table 6.34. It is difficult to see any evidence of differential response patterning between the groups. The most notable points are:

i) All groups show a high positive respiration rate response to a stress of 2 cm. water pressure.

ii) The normal subjects show consistently less response to the stresses in the second session (except for the respiration rate response to the 8 cm. water pressure). None of the other groups show quite such a consistent adaptation.

iii) One can see confirmed visually the overall increase in response as the airways resistance increases.

b. Basal level patterning:

Standardised scores were derived from the basal levels analysis of variance in an identical manner to the response patterns. The group responses are shown graphically in Figure 6.10. It can be seen that the neurotics have a higher heart rate in both sessions. The normal group showed a decrease in all basal levels between sessions, but the neurotic and asthmatic groups only showed a decrease in skin conductance and heart rate; respiration rate and ventilation volume stayed the same or increased on the second occasion of testing.

TABLE 6.34

STANDARDIZED RESPONSES TO STRESSORS

	Skin Conductance		Heart Rate		Respiration Rate		Ventilation Volume	
	mean	s.d	mean	s.d	mean	s.d	mean	s.d
Asthmatics/Session 1								
2 cm. H20	46.3	9.3	46.0	10.3	54.6	11.9	47.7	9.6
4 cm. H20	47.2	8.0	48.4	10.3	51.2	12.1	50.2	11.2
8 cm. H20	53.9	11.9	52.5	11.3	51.2	12.6	59.2	16.9
Session 2								
2 cm. H20	43.8	8.7	45.7	8.9	56.0	11.3	47.6	10.3
4 cm. H20	45.0	8.4	49.9	7.0	52.1	12.2	47.4	9.9
8 cm. H20	53.4	13.5	51.0	9.5	51.7	12.2	51.3	13.3
Neurotics/Session 1								
2 cm. H20	49.3	17.2	50.1	8.0	54.1	18.7	41.9	10.3
4 cm. H20	45.4	6.2	48.2	12.5	44.0	22.7	46.9	12.5
8 cm. H20	54.9	12.8	52.5	12.1	43.8	13.3	56.5	22.2
Session 2								
2 cm. H20	50.6	15.5	49.8	11.3	55.5	10.6	46.3	15.3
4 cm. H20	49.2	7.1	51.2	10.6	51.3	9.9	50.6	20.1
8 cm. H20	54.0	21.8	55.2	13.0	47.5	15.0	52.8	17.9
Normals/Session 1								
2 cm. H20	47.0	9.2	48.3	10.8	54.3	10.3	48.4	11.5
4 cm. H20	52.1	11.1	49.1	9.7	53.1	8.0	47.3	11.9
8 cm. H20	55.1	12.0	53.9	6.3	47.8	9.7	54.2	11.9
Session 2								
2 cm. H20	46.9	5.5	46.0	6.5	52.8	9.13	46.2	8.17
4 cm. H20	48.4	13.7	49.5	4.7	53.5	12.6	43.5	5.8
8 cm. H20	51.1	6.7	53.1	4.8	53.1	10.2	51.5	9.7

8. Individual physiological specificity

The study of the reliability of individual physiological measures in Section 6 of this chapter showed that basal levels are repeatable and response measures less so. It is also possible that individuals' patterns of physiological activity will show consistent difference; however, these will have to be interpreted in the light of the already proven lability of their component measures.

The standardised data obtained from the investigation of group basal level and response patterns were used for this study of individual patterning. Individual standardised values of the responses and basal levels were available and it was thus a simple matter to rank each person on the activity or reactivity in different physiological measures on any one occasion.

It was decided to compare the basal levels and responses obtained on two occasions, one in each session. The basal level measurements used were the "Pre-8 cms. stress" values from the preceding analyses of variance of basal levels; the responses were those to the 8 cm. H_2O stressor. Thirty-one asthmatic, fifteen neurotic and sixteen normal subjects had complete data with measures for all four variables in both sessions, and only the data from these subjects were incorporated in the analyses which follows.

The initial question to be answered was: does a significant proportion of these individuals show specificity? To investigate this, the sum of the squared differences between the two sets of rankings of the physiological measures was computed for each subject. For example,

if the rank order of responses on the two occasions of testing were -

	First Session	Second Session
1 :	Heart Rate	Skin Conductance
2 :	Skin Conductance	Ventilation Volume
3 :	Ventilation Volume	Heart Rate
4 :	Respiration Rate	Respiration Rate

then the sum of squared differences,

$$\Sigma d^2 = 1^2 + 1^2 + 2^2 + 0^2 = 6.$$

With a larger number of variables, it would be possible to derive r_s, the Spearman rank correlation coefficient, and assess its significance in the usual way for each individual. However, for such a small number of variables, the probability of a correlation coefficient of 1 is just equal to 0.04, and counting the number of individuals with significant correlations would be a relatively insensitive method of treating the data. An alternative method of examining the results of a group of subjects was therefore derived from the following reasoning:

If there is <u>no</u> response specificity, then the distribution of rank correlation coefficients (or their equivalent Σd^2) in a group of subjects will resemble the chance distribution, which can easily be calculated. If the group as a whole shows physiological specificity, then the distribution will:

a) be skewed towards low values of Σd^2 (i.e. a high correlation)

b) be significantly different from the chance distribution of Σd^2.

The latter hypothesis can be tested by the use of the Kolmogorov-Smirnov one sample test for goodness of fit (Siegel, 1956). This test is sensitive to any departure from the expected distribution, and a significant result could indicate a skewness in either direction. However, visual inspection of the distribution should indicate whether a significant result is due to a skewness in the predicted direction, or otherwise.

The results for the three groups are shown in Tables 6.35-6.37 (basal levels), and in Tables 6.38-6.40 (responses). All three groups of subjects show specificity of <u>levels</u> ($p<0.01$), but only the asthmatic group shows specificity of <u>responses</u>. To examine the possibility that the two forms of specificity might be associated, each group of subjects was dichotomised into approximately equal "high specificity" and "low specificity" sub-groups, and a chi-square test was performed on the resulting contingency table. It can be seen (Table 6.41) that there is no association between showing specificity of levels and of responses.

9. <u>Group differences in physiological patterning</u>

To examine the possibility that there are differences in the patterns of a group as a whole, a measure of the overall response pattern for each group was obtained. This was done by adding the rank of each physiological variable (1,2,3 or 4, with one denoting the maximum reactivity) of each subject in the group to give an

TABLE 6.35

Stability of basal level patterns Asthmatics (n=30)

Σd^2	0	2	4	6	8	10	12	14	16	18	20
Cumulative Frequency Distribution	4	8	11	18	23	26	27	27	27	29	30
Percentage Distribution	12	27	37	60	77	87	90	90	90	97	100
Theoretical Distribution	04	17	21	38	46	54	62	79	83	96	100
Differences	09	10	16	22	31	33	28	21	07	01	00

Kolmgorov-Smirnov test d max = 33 $p < 0.01$

TABLE 6.36

Stability of basal level patterns Neurotics (n=15)

Σd^2	0	2	4	6	8	10	12	14	16	18	20
Cumulative Frequency Distribution	2	9	9	12	13	13	14	14	14	15	15
Percentage Distribution	13	60	60	80	87	87	93	93	93	100	100
Differences	09	43	39	42	47	33	31	14	10	04	00

Kolmgorov-Smirnov test d max = 43 $p < 0.01$

TABLE 6.37

Stability of basal level patterns Normals (n=16)

Σd^2	0	2	4	6	8	10	12	14	16	18	20
Cumulative Frequency Distribution	48	9	9	14	14	14	14	16	16	16	16
Percentage Distribution	25	56	56	87	87	87	87	100	100	100	100
Theoretical Distribution	04	17	21	38	46	54	62	79	83	96	100

Differences

Kolmgorov-Smirnov test d max = 49 $p < 0.01$

TABLE 6.38

Stability of response patterns Asthmatics (n=31)

Σd^2	0	2	4	6	8	10	12	14	16	18	20
Cumulative Frequency Distribution	3	10	14	24	26	26	28	29	30	31	31
Percentage Distribution	10	32	45	77	83	83	90	93	97	100	100
Theoretical Distribution	04	17	21	38	46	34	62	79	83	96	100

Differences

Kolmgorov-Smirnov test d max = 39 $p < 0.01$

TABLE 6.39

Stability of response patterns Neurotics (n=15)

Σd^2	0	2	4	6	8	10	12	14	16	18	20
Cumulative Frequency Distribution	4	5	6	7	10	10	12	12	13	14	15
Percentage Distribution	28	33	40	47	67	67	80	80	87	94	100
Theoretical Distribution	04	17	21	38	46	54	62	79	83	96	100
Differenced	24	16	19	09	21	13	18	1	4	2	0

Kolmgorov-Smirnov test d max = 24 (N.S)

TABLE 6.40

Stability of response patterns Normals (n=16)

Σd^2	0	2	4	6	8	10	12	14	16	18	20
Cumulative Frequency Distribution	1	4	4	5	5	7	8	10	11	16	16
Percentage Distribution	6	25	25	31	31	44	50	63	69	100	100
Theoretical Distribution	4	7	21	38	46	54	62	79	83	96	100
Differenced	2	8	4	7	15	10	12	16	14	14	4

Kolmgorov-Smirnov test d max = 16 (N.S)

TABLE 6.41

Association of basal level and response specificity

	High basal specificity	Low basal specificity	
High response specificity	15	12	27
Low response specificity	21	13	34
	36	25	61

X^2 (with Yates' Correction) = 0.05 (N.S)

overall total. The distributions of ranks and the overall reaction patterns are shown in Table 6.42 (basal levels) and Table 6.43 (response patterns).

It is notable that although it was demonstrated that a significant number of individuals show physiological specificity, the group physiological patterns are not so stable, with the asthmatics' level patterns showing a complete reversal on the second session. In addition, the asthmatic group's correlation of -0.2 for responses does not show much evidence of stability.

There does not appear to be any one simple method of testing the hypothesis that the differences in these group physiological patterns could have arisen by chance. One approach which was tried involved extracting the number of individuals in each group who responded most (or were most active) in each physiological measure. A chi-square test could then be done to test the null hypothesis that the distribution in the three groups is equivalent. However, more than 20% of the cells had expected values of 5 or less; under which conditions the chi-square test is invalid. Nevertheless, various points can be made about the data contained in Tables 6.42 and 6.43:

a) The data for levels confirm the finding by analysis of variance that the neurotic group's heart rate is significantly higher than that of the normals.

TABLE 6.42

Frequency of distribution of basal level patterns

	Session 1					Session 2					
Asthmatics	**Frequency of rank**				**Total**	**Frequency of rank**				**Total**	**Overall patterns**
	1	2	3	4		1	2	3	4		
SC	9	7	8	6	71	6	5	9	10	83	154
HR	8	11	5	6	69	7	5	5	13	84	153
RR	8	3	9	10	81	10	10	5	5	65	146
VV	5	9	8	8	79	7	10	11	12	68	147
Between sessions correlation $r_s = -1.0$											
Neurotics											
SC	0	3	6	6	48	1	2	2	10	51	99
HR	8	2	2	3	30	5	6	4	0	29	59
RR	4	5	2	4	36	5	3	3	4	36	72
VV	3	5	5	2	36	4	4	6	1	34	70
Between sessions correlation $r_s = 0.95$											
Normals											
SC	7	6	1	2	30	6	4	0	6	38	68
HR	4	2	1	9	47	4	4	3	5	41	88
RR	5	3	5	3	38	5	4	4	3	37	75
VV	0	5	9	2	45	1	4	9	2	44	88
Between sessions correlation $r_s = 0.6$											

Each entry represents the number of subjects in the group in whose individual patterns the physiological variable are ranked 1st, 2nd, 3rd, or 4th in the relative amount of activity shown. The between sessions correlation is an estimate of the consistency of the group patterns. The total is an estimate of the group pattern, derived from the formula.

Total = (1 x no. of individuals ranked 1) + (2 x no. of individuals ranked 2) + (3 x no. of individuals ranked 3) + (4 x no. of individuals ranked 4).

TABLE 6.43

Frequency distribution of response patterns

	Session 1					Session 2					
Asthmatics	**Frequency of rank**				**Total**	**Frequency of rank**				**Total**	**Overall pattern**
	1	2	3	4		1	2	3	4		
SC	7	6	9	9	82	9	3	10	9	81	163
HR	4	7	8	12	90	5	15	8	3	68	158
RR	7	9	8	7	77	7	6	5	13	86	163
VV	13	9	6	3	61	10	7	8	6	72	131
Between session correlation $r_s = -0.2$											
Neurotics											
SC	6	3	3	3	33	3	3	4	5	41	74
HR	4	4	4	3	36	5	6	2	2	31	67
RR	2	3	4	6	44	1	5	4	5	43	87
VV	3	5	4	3	37	6	1	5	3	35	72
Between session correlation $r_s = 0.4$											
Normals											
SC	6	3	4	3	36	3	3	4	6	45	81
HR	4	9	2	1	32	5	6	3	2	34	66
RR	0	1	5	10	57	6	6	3	2	32	89
VV	6	3	5	2	35	2	1	7	6	49	84
Between correlation $r_s = 0.8$											

Each entry represents the number of subjects in the group in whose individual patterns the physiological variables are ranked 1st, 2nd, 3rd, or 4th in the relative response magnitude. The between sessions correlation is an estimate of the consistency of the group patterns. The total is an estimate of the group pattern, derived from the formula.

Total = (1 x no. of individuals ranked 1) + (2 x no. of individuals ranked 2) + (3 x no. of individuals ranked 3) + (4 x no. of individuals ranked 4).

b) Asthmatics respond most to the stressor in ventilation volume, with respiration rate and skin conductance equal third; the tendency in the normal subjects is to show most response in heart rate and skin conductance, while neurotics show most response in heart rate, with skin conductance response ranking third. Thus there is a tendency for the asthmatics to show maximal response in respiratory variables, with other subjects showing more response in non-respiratory ones.

c) There is no explanation for the inconsistency in the asthmatic group's basal level patterns, where the correlation between the two sessions is negative (and significant).

In summary, therefore, it cannot be said that there is any conclusive evidence that asthmatics show different patterns of physiological functioning when compared with non-asthmatic groups.

10. <u>The relationship of psychological and psychophysiological measures</u>

It is fairly apparent from the results presented above that the psychophysiological measures used in thus study do not differentiate the experimental groups to any marked degree. In particular, there is scanty evidence to suggest that asthmatics respond more to the respiratory stressor used here than do the non-asthmatic groups;

yet there is some evidence that the physiological measures are consistent within individuals, and it therefore makes sense to try to relate these measures to the psychometric measures of personality which were also collected from these subjects. Information is available on two quite different dimensions of physiological activity for each individual. These are -

a) Measurements of the response to stress and basal levels in each of the physiological variables.

b) An assessment, for many of the individuals in the study, of the consistency of their physiological functioning patterns.

The review of literature pointed out that many workers have tried to relate responsiveness to personality, but with variable and inconclusive results. The method of choice to explore any possible interrelationships between personality traits and psychophysiological measures is factor analysis, as used in the studies cited previously by Berger & Fahrenberg & Delius (op cit.). However, Berger was content merely to establish patterns of psychophysiological measure and then to attempt an interpretation if they were cross-validated in his two samples. Armstrong and Soelberg (1968) have discussed the interpretation of factor-analytic data, and provide trenchant criticisms of the many reports of factor analyses which do not attempt to measure factor reliability. One approach to this problem is to set up hypotheses beforehand. The ones to be adopted here are -

a) It is highly probable that the analyses will yield factors loading on the measures of anxiety and neuroticism, and introversion-extraversion. It is hypothesised that the loadings of psychophysiological variables on these factors may differ in the asthmatic and non-asthmatic groups.

b) Any evidence of generalised arousal will be found in a factor which loads on the basal level measurements of all variables.

It was further decided only to attempt interpretation of factors that appeared in the factor-analyses of both the asthmatic and non-asthmatic groups' data.

The factor-analyses were carried out using the BMD 72X Factor Analysis program (Dixon, 1965). In accordance with standard practice, a preliminary principal components analysis for each matrix of inter-correlations was performed, to establish the number of latent roots greater than one. Kaiser (1960) recommends this as the best estimate of the number of factors to be extracted, on the grounds of algebraic necessity, psychometric reliability and psychological meaningfulness. A principal factors solution was then obtained, and this was rotated to both oblique (simple) structure, and to orthogonal structure, using the varimax and bi-quartimin analytical solutions, respectively (Harman, 1967).

Because of the relatively small numbers in the two control groups, and because the main objective of the exercise was to investigate any differences between the asthmatic and non-asthmatic groups, the normal and neurotic subjects were treated as one group of controls to be

compared with the asthmatics. The following variables were included in the analysis:

1) Basal skin conductance, heart rate, respiration rate, ventilation volume (the "pre-8 cm." value).
2) The response to the 8 cm. H_2O pressure stressor (in skin conductance, heart rate, respiration rate, ventilation volume) in session one.
3) TMAS: total score.
4) EPI: N and E scales.
5) HDHQ: Total Hostility and Direction of Hostility scores.
6) 16 PF : all 16 first-order factors.

The correlation matrices for the two groups and the factor loadings can be found in Appendix 4. Table 6.44 summarises the significant correlations that were noted in each matrix.

The initial principal components analyses of the two correlation matrices showed that there were 10 latent roots greater than 1.0 in the asthmatics' correlation matrix, and nine in the controls' matrix. However, the computer program gave meaningless results when ten factors were extracted, so the number was reduced to six, which was the maximum number found empirically to give an algebraically meaningful solution.

In interpreting the correlations and any subsequent factors in the control group's correlation matrix, it should be borne in mind that these may not necessarily imply significant correlations of the two measures <u>within</u> each of the two groups. For instance, it is possible that the correlation between 16 PF factor B (intelligence), and basal

TABLE 6.44

Significant correlations between physiological and psychological measures ($p < 0.05$)

Physiological	Psychological
A). Asthmatics	
Basal Heart rate	HDHQ (Direction)
Basal respiration rate	16PF :F(-), G (+), L (+)
Respiration rate response	TMAS, HDHQ (total), 16PF :C (-1, L (+)
B). Non-Asthmatics	
Basal skin conductance	16PF : B (+)
Skin conductance response	16PF : A (+), C (+), G (-), O (-)
Basal heart rate	TMAS, EPI (N scale), EPI (E scale)
	HDHQ (total + direction)
	16PF : N (-), Q_3 (+)
Heart rate response	16PF : N (-)
Basal respiration rate	TMAS, 16PF : I (+). Q_1 (+)
Respiration rate response	16PF : I (-), L (-), O (-)
Basal ventilation volume	TMAS, 16PF : B (-)
Ventilation volume response	EPI (E scale)

skin conductance, is due to the significant differences reported in Chapter 5 between normals and neurotics on factor B, and the difference in skin conductance level (which was not significant) between the two groups. Similarly, the correlations between the respiration rate response and some of the psychological measures may reflect only the fact that neurotics showed a lower response than normal subjects and scored higher on 16 PF factors L and O. However, since most of the physiological variables did not differentiate between groups, most of the significant correlations may be said to reflect a genuine "within-the-whole-control-group" relationship, rather than a spurious "between-control-groups" correlation. Storms (1958) and Creasy (1959) have discussed this problem in more detail. One of the assumptions underlying this analysis is than any psychophysiological relationships are similar in normal and neurotic groups, so that the between-group and within-group dimensions of covariation will be equivalent. What this analysis is designed to do is to look for differences between a mixed group (in terms of anxiety and neuroticism) of non-asthmatics and a heterogeneous group of asthmatics.

In assessing the significance of the factor loadings on each variable, the recommendation of Harman (1967) was followed, whereby factor loadings greater than the average correlation in the matrix are considered to be significant. The method of Kaiser (1968) was used to assess the average correlation in each of the two matrices. It is computed from the formula:

$$\text{average correlation} = \frac{\lambda - 1}{p - 1}$$

where λ = largest latent root in a principal components analysis of the matrix

p = number of variables (in this case, 29).

The average correlation of the asthmatics' correlation matrix was 0.15, and that of the control group's correlation matrix was 0.23.

11. Discussion of the factor analysis

Table 6.45 shows the factor loadings for each of the two matrices, with both the orthogonal and oblique rotations. A comparison of the orthogonal and oblique solutions showed that they were very similar. For the asthmatics, the first four factors were equivalent, the 5th orthogonal factor was equivalent to the 6th oblique one, and the 6th orthogonal factor was equivalent to the 5th oblique one. For the control group's matrix, the first two factors were equivalent, the 3rd orthogonal one was equivalent to the 4th oblique one, the 4th orthogonal to the 6th oblique, the 5th orthogonal to the 5th oblique and the 6th orthogonal factor to the 3rd oblique one. Thus it is only necessary to consider one set of results, and the following discussion will be confined to the results of the oblique rotation.

For both groups, the first factor is clearly an anxiety factor, since it loads heavily on the TMAS, EPI 'N' scale, and the 16 PF first order traits that comprise the second order anxiety factor. What is of note are the physiological measures that load on this factor in each group. For the asthmatic, respiration rate response and ventilation volume response show significant loadings, while in the controls heart

TABLE 6.45

Summary of Factor Matrices

	Asthmatics						Non-Asthmatics					
Factor	V1/01	V2/02	V3/03	V4/04	V5/06	V6/05	V1/01	V2/02	V3/04	V4/06	V5/05	V6/03
Variable												
SC level					O VO					O	VO	O
SC response			V		V VO					VO		V
HR level				VO			VO	VO				O
HR response			VO								VO	
RR level	VO		VO	VO				VO		VO	O	
RR response	VO		VO	VO				VO				
VV level				VO			V	VO		VO		
VV response	VO		VO					VO			VO	
TMAS	VO	V		VO		O	VO					
EPI - N	VO	VO	VO				VO					
EPI - E		VO						VO	VO	VO	V	
HDHQ - Total	VO	VO			V		VO					
HDHQ - Direc.	VO	VO	VO		VO VO		VO		VO			
16PF A		VO			VO					VO		
B					VO VO		VO			VO		
C	VO						VO					
E	V	VO			O VO		O		VO			VO
F		VO		VO	VO				VO			
G				VO	O VO		VO			VO	VO	
H	VO	VO					VO	V	VO	VO	O	O
I	VO	VO	V	VO	VO VO			VO				
L	VO	VO	O	VO	V		VO		VO			O
M	VO			VO	O VO				VO		VO	
N				VO	V VO			VO			VO	VO
O	VO	VO					VO			V		
Q_1			VO		VO		VO	VO				VO
Q_2		VO			VO					O		
Q_3	VO						VO					
Q_4	VO						VO		VO			

V - indicates significant loading of varimax factor on variable

O - indicates significant loading of oblique factor on variable

rate is the main physiological measure (with basal ventilation volume showing a nearly significant loading). The 'coefficient of similarity' (Barlow and Burt, 1952) between these two factors was calculated to be 0.91.

The only other factor which is similar in the two groups, and can be named is represented by factor 2 in the asthmatic group and factor 4 in the non-asthmatic group. This loads heavily on the EPI extraversion scale, HDHQ Direction of Hostility, and 16 PF traits E, F and H (which are included in Cattell's second order introversion-extraversion factor). In the asthmatic group, 16 PF traits A and Q_2 also load on this factor. These two 16 PF traits also load on Cattell's second order introversion-extraversion factor -- thus it is reasonable to identify this as a general introversion-extraversion factor. The coefficient of similarity between the two was 0.80. It can be seen that none of the psychophysiological measures load on this factor at all.

An examination of the remaining factors shows that none of them can be matched across the two groups. In particular, there is no factor which reflects any generalised physiological arousal (i.e. positive loadings on all the physiological basal level variables), though in contrast, non-asthmatic factor 6, which loads on skin conductance response, respiration rate response and ventilation volume response, and asthmatic factor 3, loading on heart rate response, respiration rate response and ventilation volume response, could be considered measures of general physiological responsivity. However,

the loadings of the various psychological tests on these two factors are completely different.

12. The personality correlates of response specificity

To examine the possible relationship of personality traits to response and basal level specificity, each group of subjects --- normals, neurotics, and asthmatics --- were divided into roughly equal sub-groups, one division contrasting individuals with high and low basal level specificity (based on each individual's value of d2), the other dividing them into two groups with high and low response specificity. The median value of Σd^2 was taken as the cutting point in each group. The mean score on each of the personality scales used in the preceding factor analysis was computed for each subgroup. These are shown in Tables 6.46 and 6.47.

In the division by levels the most notable observation is the consistently higher scores in 16 PF Factors O and Q_4, of the low specificity sub-group in all three main groups of subjects. It will be recalled that these two traits comprise two of the components of the 16 PF second order anxiety factor. However, the TMAS score does not show such consistent differences between the groups. An analysis of variance was performed to test the significance of the differences between the high and low specificity sub-groups. The method of unweighted means (Winer, 1962, p.242) was used, since there were unequal numbers in the 6 cells (3 groups x 2 levels of specificity). The results are shown in Table 6.48, from which it emerges that the differences are indeed significant. A note of caution is in order

TABLE 6.46

PSYCHOMETRIC TEST SCORES OF SUBGROUPS DIFFERING IN BASAL LEVEL SPECIFICITY

Scale	Asthmatics				Neurotics				Normals			
	High		Low		High		Low		High		Low	
	mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d
TMAS	16.7	9.7	18.0	5.8	28.7	9.9	27.5	7.9	6.6	6.6	14.9	8.2 *
EPI												
N	12.0	5.7	12.5	6.2	16.7	4.8	15.3	2.5	5.4	3.8	8.6	6.0
E	10.4	3.6	9.3	5.0	12.3	4.0	11.0	3.7	9.6	3.8	9.7	1.9
HDHQ												
Total	16.8	6.4	17.0	6.7	25.0	12.2	25.5	3.1	9.2	5.6	14.1	9.1
Direction	2.3	6.4	4.5	7.4	3.7	4.8	-1.8	8.8	-1.0	2.5	1.6	5.8
16PF												
A	4.9	1.2	4.9	2.2	5.4	1.5	5.3	1.9	4.6	1.6	4.0	2.1
B	8.3	1.8	7.8	2.2	6.6	2.1	7.0	2.5	8.0	1.5	9.3	1.1
C	5.3	1.8	4.8	1.7	3.4	2.4	3.0	1.1	6.2	2.5	5.0	1.2
E	4.7	2.1	4.8	2.4	5.8	1.9	6.8	1.8	4.2	1.6	5.9	2.0
F	5.5	2.1	5.3	2.5	6.1	1.5	4.8	2.8	6.3	2.0	5.4	1.5
G	4.9	1.8	5.1	2.5	5.1	3.0	5.0	1.8	4.9	1.2	4.7	2.0
H	4.8	1.9	4.3	2.2	5.2	2.4	4.0	1.8	5.3	1.7	4.9	2.0
I	4.7	2.2	4.8	1.3	4.0	1.6	4.7	1.8	5.7	1.5	4.9	1.8
L	5.8	1.9	5.5	1.8	6.9	2.1	7.0	1.3	5.1	2.3	4.9	2.0
M	5.5	2.3	5.1	1.9	6.4	1.5	5.8	1.2	5.9	2.2	6.9	1.3
N	5.2	1.5	5.3	2.2	4.6	2.3	5.7	1.9	5.2	1.1	5.9	1.9 *
O	5.2	2.1 *	5.8	1.3	7.0	2.6	8.8	1.2	4.1	2.2	6.7	1.1 *
Q1	7.2	1.9	6.3	2.1	6.9	2.8	6.3	1.2	7.0	1.7	7.1	2.2
Q2	6.3	1.8	4.7	2.2	5.4	1.7	6.3	2.3	5.9	1.1	6.6	1.5 *
Q3	5.5	2.1	5.2	1.2	4.6	2.2	4.8	2.1	7.0	2.0	5.0	1.6 *
Q4	5.3	1.9	6.6	1.4	7.4	1.6	8.0	1.3	4.7	2.5	5.7	1.4

* Difference between sub-groups significant at $p < 0.05$ (t test)

TABLE 6.47

PSYCHOMETRIC TEST SCORES OF SUBGROUPS DIFFERING IN RESPONSE SPECIFICITY

Scale	Asthmatics				Neurotics				Normals			
	High		Low		High		Low		High		Low	
	Mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d
TMAS	15.8	7.8	17.7	8.7	32.1	7.2	24.8	9.2	7.6	6.4	12.8	9.5
EPI												
N	11.5	5.6	12.4	6.1	17.7	3.4	14.8	4.1	5.1	4.5	8.5	5.1
E	10.2	4.3	9.9	4.1	12.3	4.6	11.4	3.3	9.1	3.4	10.1	2.6
HDHQ												
Total	16.8	6.6	16.8	6.2	29.0	7.8	22.1	10.1	8.3	3.2	14.6	9.4
Direction	3.8	8.3	2.5	5.5	3.3	3.6	-1.3	9.0	3.8	3.9	-1.3	4.9*
16PF												
A	4.9	1.5	4.9	1.7	5.3	0.8	5.5	2.1	4.9	1.6	3.8	1.9
B	8.1	2.0	8.2	1.9	7.3	2.3	6.3	2.2	8.8	1.1	8.4	1.8
C	4.9	1.9	5.4	1.6	2.6	0.8	3.9	2.4	6.9	1.7	4.5	1.7*
E	4.5	1.9	4.9	2.4	5.1	1.7	7.1	1.6*	4.5	1.9	5.4	1.9
F	5.9	2.2	5.2	2.2	5.3	2.2	5.1	2.1	5.6	1.5	6.3	2.1
G	4.3	1.6	5.3	2.3	4.0	2.0	6.0	2.7	4.9	2.0	4.8	1.0
H	4.2	2.0	4.9	2.0	3.9	2.2	5.5	2.1	5.6	1.8	4.6	1.8
I	4.6	2.4	4.8	1.4	3.6	1.7	4.9	1.4	4.9	1.7	5.8	1.5*
L	5.8	2.1	5.6	1.6	7.1	2.1	6.8	1.5	3.9	1.4	6.1	2.2
M	5.8	2.2	5.1	2.0	6.3	1.1	6.1	1.6	7.1	1.3	5.5	2.1
N	5.5	1.9	5.0	1.7	4.7	2.1	5.3	2.2	5.3	1.2	5.8	1.8
O	6.1	1.6	4.9	1.8	7.9	2.2	7.6	2.6	4.4	2.3	6.1	1.8
Q1	7.4	1.6	6.6	2.2	5.9	2.3	7.4	2.1	6.9	2.0	7.3	1.9
Q2	5.2	2.1	5.9	2.0	6.6	2.0	5.1	1.8	5.9	6.4	6.5	1.7
Q3	5.4	2.1	5.6	1.7	4.1	2.2	5.1	2.1	6.3	2.0	6.0	2.3
Q4	6.5	2.1	5.4	1.3	7.4	1.7	7.9	1.3	4.1	1.8	6.1	2.0

* Difference between sub-groups significant at $p < 0.05$ (t test)

TABLE 6.48

Comparison of the psychological test scores of high and low basal specificity sub-groups

16PF Factor O

Source of variance	df	SS	MS	F
Specificity	1	37.44	37.44	10.20***
Experimental Groups	2	73.42	36.71	10.00***
Interaction	2	9.30	4.65	1.27 (N.S)
Error	55	201.90	3.67	
Total	60			

16PF Factor Q_4

Source of variance	df	SS	MS	F
Specificity	1	12.54	12.54	4.04*
Experimental Groups	2	58.92	29.46	9.50***
Interaction	2	1.13	0.57	1 (N.S)
Error	55	170.58	3.10	
Total	60			

regarding this analysis, as the measure of specificity (or consistency) is relatively crude, being derived from data collected on only two occasions. These findings should therefore be regarded somewhat tentatively. No other personality factors showed such marked and consistent differences between groups.

No consistent trends were observed across the three groups of subjects when they were divided into high and low response specificity sub-groups. In view of the fact that only the asthmatics (as a group) showed evidence of response specificity, and in view of the previously demonstrated unreliability of these responses when they were considered individually (Section C.6), this data will not be considered any further.

Tables 6.49 and 6.50 show the mean scores of the high and low specificity sub-groups on the psychophysiological, basal level and response measures. It can be seen that there is no specific trend in the results; thus, one can conclude that the procedure used to assess specificity in individuals is not dependent in any way on the absolute magnitude of levels and responses.

D. Discussion

The discussion of all the findings in this chapter will be deferred to the next, and final chapter, where an attempt will be made to integrate and summarise all the findings obtained in this research.

TABLE 6.49

Basal level and response measurements of sub-groups differing (in basal level specificity)

Measures	Asthmatics				Neurotics				Normals			
	High		Low		High		Low		High		Low	
	mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d
SC level	815	199	886	207	777	286	715	107	844	67	837	87
SC response (log u mhos x 10^4)	23	47	4	16	26	44	15	32	35	42	7	17
HR level	84.0	17.0	82.0	14.9	94.0	19.7	83.2	18.9	73.8	9.5	76.1	10.1
HR response (beats/min)	2.4	3.6	5.8	4.7*	2.4	4.6	3.2	3.3	2.8	3.3	4.3	1.1
RR level	13.6	5.9	13.4	4.2	15.5	6.9	15.0	2.8	14.6	4.4	14.0	5.4
RR response (breaths/min)	0.6	4.1	0.3	2.5	1.3	3.7	1.3	2.8	2.7	2.9	0.7	1.7
VV level	7.2	2.7	6.3	1.6	8.9	4.9	5.8	0.8	6.0	1.1	5.9	2.4
VV response (litres/mm)	3.9	3.2	1.6	1.3*	4.3	4.6	1.3	2.3	1.3	1.4	3.8	1.8*

* = Significant difference between high and low specificity group ($p < 0.05$. t test)

TABLE 6.50

Basal level and response measurements of sub-groups differing (in response specifity)

Measure	Asthmatics				Neurotics				Normals			
	High		Low		High		Low		High		Low	
	mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d	mean	s.d
SC level	879	181	813	207	863	219	656	199	838	89	844	60
SC response (Log u mhos x 10^4)	10	27	24	48	14	26	29	47	24	35	22	37
HR level	85.0	15.2	81.7	16.4	101.3	13.9	79.6	18.5*	73.6	9.2	76.0	10.2
HR response (beats/min)	1.9	2.8	1.4	4.9	3.3	3.3	2.3	4.7	4.6	2.0	2.3	2.7
RR level	11.5	5.5	14.9	4.4	14.7	6.5	15.9	4.9	15.9	4.6	12.8	4.5
RR response (breaths/minute)	0.5	2.1	0.1	4.3	0.6	4.4	2.0	1.5	1.4	1.8	1.6	3.1
VV level	6.8	3.0	6.8	1.8	6.9	3.1	8.4	4.8	6.5	2.0	5.4	1.4
VV response (litres/mm)	2.7	1.5	3.1	3.5	4.0	5.7	2.3	3.0	2.2	1.9	1.9	1.8

* = Significant difference between high and low specificity group ($p \leq 0.05$, t test)

CHAPTER 7

CONCLUSIONS AND GENERAL DISCUSSION

A. Summary of Principal Findings

1. Groups of normal and neurotic subjects could not be differentiated on two psychophysiological tests, habituation of the psychogalvanic reflex and forearm blood flow. This initial finding, by casting doubt on the validity of the tests, made it impossible to draw any firm conclusions about the nature and extent of anxiety in the asthmatic group, as assessed by these methods.

2. The asthmatic group did not differ from the normal control group on the TMAS, but scored significantly higher than the normal group, and significantly lower than the neurotic group, on the EPI Neuroticism scale. No differences between the asthmatic and normal groups were noted on the HDHQ or 16 PF Questionnaire.

3. When compared with published normative data, the asthmatics were more neurotic and introverted on the EPI. On the 16 PF they were found to score lower on factors C,E,I and N and higher on factors Q1 and Q4.

4. Psychophysiological measures were recorded on two occasions from the subjects while they were relaxed and while exhaling against an airway resistance. A preliminary analysis showed that significant responses were obtained in all physiological variables.

5. Several different response measures were computed, and correlation coefficients calculated between them all and with pre-stress basal level measurements. High correlations were found between these different response measures, and with the exception of respiration rate responses, insignificant correlations were obtained with any basal level measurements.

6. Only basal level measurements of heart rate differentiated between the normal and neurotic groups. The asthmatics did not differ from any of the other groups on any basal level measurements.

7. An analysis of the response measures showed that significant differences between responses to the different stressors were obtained in all physiological measures. The only significant difference between the groups found was that neurotic subjects consistently responded to the stressor with a larger decrease in respiration rate than was observed in the other two groups. No interaction between groups and stressors was observed.

8. There was a high concordance between measurements of basal level taken at different times in the same session. Heart rate, respiration rate and ventilation volume measurements from the two sessions were also highly correlated, but SC level measurements showed no such consistency across sessions.

9. The consistency of the responses to the different stressors within sessions was less reliable, with only respiratory measures showing any marked evidence of individual consistency. Similar results were obtained when responses from the two sessions were compared.

10. The response and basal level measurements were standardised to permit comparisons between measurements of different physiological variables. The normal group showed a consistent adaptation of response and basal level measurements in all four variables between the two sessions; however, this pattern was not observed in the other two groups. There was little evidence of response patterns unique to each group.

11. Individual response patterns were examined to determine if individuals showed any consistency of physiological functioning patterns. Level patterns were consistent in all three groups, but only the asthmatic group showed consistent response patterns.

12. A ranking technique was used to assess the most common group physiological functioning patterns. There was a tendency for the asthmatics to show more activity in respiratory measures than non-respiratory ones.

13. A factor analysis was performed incorporating both the measures of personality and psychophysiological measurements of level and response. The principal finding was that both respiration rate response and ventilation volume response were linked to measures of anxiety in the asthmatic group, but the only measure that was found to correlate with anxiety in the control groups was heart rate. A factor identified as an introversion-extraversion factor did not lead on any psychophysiological measurements.

14. The personality traits of subgroups showing high or low specificity of basal level and response patterns were examined. Individuals in all sub-groups showing low basal level specificity scored higher on 16 PF factors O and Q4 than those showing high basal level specificity. No similar tendencies were found when groups of individuals differing in response specificity were compared.

B. A Discussion of Some Further Factors Which May Influence the Results of the Breathing Study

At first sight, it might seem disappointing to be confronted with page after page of evidence that asthmatics are no different from the two control groups in the magnitude of their physiological responses to a respiratory stressor. Yet this broad conclusion can be taken as a confirmation of the belief that any interpretation of psychophysiological measurements must be considered in the context of the psychological processes of which they are a part. Of these processes, the most important are probably the cognitive mediating processes leading to the appraisal of a threatening situation, and it is unfortunate that there is no data available to be reported here on this aspect of the study.

Before the implications of the results are examined in detail, it may be worth considering some of the more basic factors that could have influenced them.

1. Are psychophysiological measures independent?

It is misleading to try to compare the reactivity of different physiological systems if in fact they are not functionally independent. There is some evidence that certain of the psychophysiological measures

used in this study are functionally related. Thus, Engel and Chism (1967) found that a 20% increase or decrease in respiration rate sustained for ten minutes did not change average heart rate in a group of subjects, but increases in respiration rate decreased the standard deviation of heart rate, and decreases in respiration rate increased it. However, the subjects considered individually showed wide (and significant) differences in their results. No attempt was made to control for differing ventilation volumes resulting from the separate experimental conditions. Schneider (1930) reported that 80% of subjects showed an increase of heart rate over a period of five minutes while hyperventilating. Brown (1953) suggested that hyperventilation would cause a vasoconstriction, which could account in part for the increase in heart rate observed by Schneider. The vasoconstriction might also affect the electrodermal properties of the skin near the blood vessels.

The crucial point that emerges from all these findings is that there are marked <u>individual</u> differences in the inter-relationships between variables; thus the problem of non-independence of physiological measures is important to the extent that it may affect observations of individuals. This is tantamount to a restatement of the concept of individual response specificity, but it also carried the implication that <u>group</u> differences in physiological patterning cannot be readily explained by universal physiological "laws".

In this study, a significant correlation was obtained between heart rate response and ventilation volume response to the 8 cm. H_2O pressure resistance in both the asthmatic and non-asthmatic groups. Furthermore, both groups show a factor in the factor analysis which loads on these two variables. Thus, in this study, individuals who show large increases in ventilation volume when stressed also show increases in heart rate. This would be expected to affect the distribution of the different response patterns obtained, such that there would be more examples of high ventilation volume + high heart rate and low ventilation volume + low heart rate patterns than would be expected.

2. The problem of stimulus specificity

Because of the peculiarly "physiological"nature of the respiratory stressor employed in this research, there may well be a preferred method of response which has little to do with any cognitive or emotional mediating processes occurring the individual. It was noted in the previous chapter that a decrease in respiration rate and a small increase in ventilation volume could be considered as an "efficient" method of breathing against the airways resistance. This is quite a reasonable supposition, since an increase in ventilation volume took place as a response to the stressor in almost all subjects, irrespective of whether they were asthmatics or non-asthmatics. The simplest explanation is that in order to open the valve during expiration, the dead space between the mask (where the pressure sensor was located) and the expiratory valve had to be filled with air at the higher-than-atmospheric pressure required to open the valve. Thus, in order to

force the air pressure to above this value, more air would have had to have been exhaled, and this would result in an increased inspiratory ventilation volume. However, more complex responses of the respiration regulating mechanisms cannot be ruled out - for instance, it is possible that there was a build up of CO_2 in the lungs, which would have stimulated ventilation. This situation is quite unlike the more psychological stressors used by other workers (e.g. Lazarus, 1967), who employed experimental situations where there were no constraints of this nature on the psychophysiological responses. However, one advantage of positing an expected response is that it provides a yardstick by which to measure deviations from that expectation.

3. Statistical considerations

The method used to standardise the physiological data imposes some limitations on the response patterns that can be obtained. The use of the mean response derived from the experimental data alone effectively means that some of the subjects must show high standardised responses while others must show low ones. Because no normative psychophysiological data collected from large numbers of subjects is available, it is not possible to compare all the experimental groups with an external criterion. If this could be done, one possible result obtained could be that all groups were shown to be hyper- or hypo-reactive. If psychophysiological measures are ever to be used in the applied context of individual assessment, the collection of data like this will be necessary, and it would also be illuminating if used in experimental studies which are designed to test specific hypotheses.

4. The effect of non-psychological variables on psychophysiological measures

No attempt was made directly to control for or assess the effects of such individual variables as age and sex on physiological measures. It is known (Malmo and Shagass, 1949) that the amount of heart rate variability (sinus arrythmia) is megatively related to age, and that female heart rate variability is higher than that found in males. Similarly, skin resistance is known to be affected by the phase of the menstrual cycle, and this could clearly have increased the variance in the SC measurements obtained for women. Since the groups of subjects in this study contained the same proportion of male and female subjects, and were age matched, these factors should not have affected the results in a biased way, but would undoubtedly have increased the error variance.

C. Discussion of the Results of the Stress Response Study

1. The relationship of response patterns to anxiety

Without doubt the most interesting finding is that in anxious asthmatics the response to stress was related to large responses in respiratory variables, while in non-asthmatics, only basal heart rate distinguished more anxious from less anxious subjects. If it can be assumed that the anxiety factor represents a dispositional measure (which is not an unreasonable assumption, since the EPI 'N' scale and the 16 PF scales are designed to be measures of trait anxiety), then this finding cannot be explained solely on the basis that feelings of anxiety in asthmatics are associated with increased respiratory

activity. This is not to deny that some asthmatics may have been quite anxious at the time of testing, but unfortunately there is no evidence to substantiate this. These results are indirectly in agreement with the findings of Selesnick, et al. (op cit.), since they found that the only effect of anxiety-reducing drugs on asthmatics was to reduce the respiratory response to stress. One could easily predict that anxious asthmatics in this study would respond identically with a similar treatment. It is also interesting to note that asthmatics differ from non-asthmatics in that their anxiety is not associated with higher heart rate, in addition to the respiratory variables. This isin complete contrast to the many findings (including the results of this study) that anxious psychiatric patients show higher basal heart rates than non-anxious subjects. It would be intriguing to know if this difference in relationship is reactive to the asthma itself, or whether constitutional differences between asthmatics and non-asthmatics present before the onset of the disorder may be responsible for it.

The fact that no specific physiological response measures were found to relate to anxiety in the non-asthmatic group may be a reflection of the lack of response specificity found in these individuals. One could surmise that in healthy individuals physiological responses will occur indiscriminately in different organ systems, but in those suffering from psychosomatic disorder the response is firmly channelled into one particular system. This is in line with the claims by Engel and Bickford (1961) that hypertensives are over-reactive to laboratory stressors (mental arithmetid and the cold pressor test) in blood

pressure, and that they respond with more consistent individual response patterns than control subjects. This would account for the finding that only the asthmatic group showed consistent response patterns. Similarly, the slight tendency for asthmatics to respond more in respiratory variables than the control group to stress is in line with the hypothesis of increased respiratory reactivity in these individuals, but this finding was extremely tentative and would require further research to confirm it.

The linkage found between trait anxiety and respiratory responsivity in the asthmatics calls for an explanation, and several attractive hypotheses could be put forward. It should be said at this point that a crude, but objective index of asthma severity (Aitken, Zealley and Rosenthal, 1971) showed no relationship to anxiety or to any of the respiratory measures used in the study. Thus one can rule out a priori the severity of the disorder as a potential factor influencing the relationship under discussion.

The most appealing explanation, and one that fits in with recent research on stress and with the phenomenology of anxiety, is that more anxious individuals will perceive many situations as threatening. If we assume that the respiratory system is indeed the most responsive physiological system in asthmatics, then the well-known relationship of threat to autonomic arousal (Lazarus, 1967) will induce large respiratory responses. However, this explanation assumes that the anxious asthmatics would have been more likely to experience greater anxiety and threat in the experimental situation. Future studies would

have to include means of assessing the subject's appraisal of the situation as threatening at the time. They would also have to employ a variety of different stressors, since this explanation assumes that the respiratory stress used here is equivalent to others in producing anxiety; and it was noted in Section B.2 of this chapter that the stressor used may have had quite stimulus-specific properties, which would have influenced the responses quite independently of any psychological mediating processes.

2. The relationship between specificity of basal level patterns and anxiety

There have been no previous reports of the personality characteristics of individuals who characteristically show stable physiological pattern specificity, and any attempt at explanation must therefore be purely speculative, to be tested subsequently by experiments designed to investigate this phenomenon explicitly.

The data in Table 6.49 demonstrated that there were no consistent differences between high and low specificity subgroups in any of the physiological measures. Thus one can rule out the explanation that more anxious individuals showed higher levels of arousal and hence higher levels of physiological activity. Indeed, 10 out of the 12 possible comparisons of the high specificity/low anxiety and low specificity/high anxiety subgroups indicate that the lower specificity subgroups show lower basal levels than the high specificity individuals.

No explanation that requires the assumption of a generalised state of arousal to be associated with anxiety will suffice by itself, since this assumption would not imply any differential changes in activity in the measures considered separately from one occasion to the other. One possible explanation is that the known association of anxiety with the short term lability of physiological measures (e.g. SC fluctuations) can be extended to longer term variations in basal levels, and that these changes are independent in different physiological measures. However, there is no evidence for these assumptions.

Another possibility is that more anxious people are subject to greater changes of mood than non-anxious ones. If this is so, then the varied physiological patterns known to be associated with specific mood states (e.g. grief (Lindemann, 1944), sadness and mirth (Averill, 1969)) would account for these differences. This explanation is as tentative as the first, but could easily be tested by repeated sampling of physiology and mood in a group of individuals.

D. Implications of the Findings for Further Research on Asthma and for the Clinical Management of Asthmatics

It was emphasised that many questions remain unanswered about the psychological mediating processes that may have been assumed to take place as part of the response to the breathing stressor. Yet some points do emerge from the study that may assist in the management of asthmatics, and should stimulate further research. One is that there were many anxious asthmatics, and since this anxiety was not related to

the severity of the respiratory dysfunction, a clear need for its separate treatment was indicated. It was also shown that these asthmatics responded to a stressor with large increases in respiratory activity, which could prove distressing to the patient and might possibly be the cause of a further increase in the level of anxiety (without necessarily assuming that this would trigger an attack of asthma). Thus, there is a clear case for treatment which attempts to break this vicious circle, and one which is suitable is behaviour therapy (Wolpe, 1966), using the technique of systematic desensitisation, which should succeed in reducing the distress. This treatment has already been shown (Moore, 1965) to improve the clinical condition of asthmatics, when judged by objective indices such as respiratory function tests.

It is equally noteworthy that as a group, the asthmatics did not show much evidence of increased anxiety and neuroticism. Although their EPI scores showed the asthmatics to be more neurotic than both the normal control group in the study and the general population, a further analysis of this data (Aitken, Zealley and Rosenthal, 1971) revealed that only the women asthmatics were responsible for this result; the men's neuroticism scores being similar to that of the normal population. Thus it cannot be concluded that higher neuroticism is reactive to the dysfunction in all asthmatics; it is more likely that neuroticism affects the declaration of symptoms. This has been found to be the case in essential hypertension, where Robinson (1963) observed that out-patients diagnosed as hypertensive had nigher neuroticism scores on the MPI than a

undiagnosed sample of individuals with high blood pressure from outside hospital. Similarly, Cochrane (1969) found that both hypertensives being treated by their G.P.s and a matched control group being treated for other reasons had a higher mean neuroticism score than a comparable group of individuals not being treated by a physician.

The results reported here probably beg as many new questions as they answer old ones. It was mentioned before that we do not know what cognitive factors intervened in the subjects' appraisal of the stressful situation, and indeed, a study of asthmatics' attitudes generally towards their disorder could be rewarding and might help to pinpoint these individuals who become hyperanxious as a response to bronchioconstriction. A prospective study of physiological functioning in a large group of healthy individuals would provide answers to the question of whether the reactivity of the respiratory system precedes or is a function of the illness. Findings that respiratory hyper-responsivity precedes the illness would be in agreement with the notion of organ weakness as a precursor of asthma. Further psychophysiological studies of asthmatics themselves are needed, to extend the present study, and especially to investigate whether large respiratory responses are elicited by other emotion-arousing stimuli. If the psychodynamic hypotheses of Alexander are correct, then the nature of the emotional feelings should be critical.

E. Some Final Thoughts on the Place of Psychophysiological Techniques in Personality Research

The perceptive reader may have noted a certain vagueness throughout this thesis in the use of such terms as "anxiety". Sometimes it has been used to describe a lasting personality characteristic, and at others a more transient state. The results in Chapter 4 - where two psychophysiological tests measuring quite specific indicants of anxiety were unable to distinguish between groups of individuals who could be described as non-anxious and anxious as a personality trait - seem to call into question the relevance of psychophysiological measures in personality research. Certainly the correlational approach, as used in this study and in many of the others cited in the review of literature cannot ultimately be more than an elaborate (though in these computer days, easy) data reduction exercise; although within a limited context, such as in this study, it may yield interesting and potentially valuable results. It is clear that psychophysiological measures have some use in the assessment of episodic variables, such as transitory mood states, but the inferences that can be made from these measurements about the relationship of psychophysiological measures to personality are indirect. To the extent that one may be investigating the differential sensitivity to situations within the same individual as a function of a given personality trait, a promising line of attack may be the ipsative approach mentioned in chapter 2. However, this approach cannot answer the more basic question of how personality may be dependent on individual biological differences.

What has been lacking in much recent psychophysiological research is any attempt at an analysis of the role of physiological activity by itself as a determinant of behaviour. For instance,* only recently (Epstein, 1967) has a systematic formulation of the relationship between anxiety and physiological arousal been propounded. In this theory, the development of anxiety is seen as an attempt by the organism to avoid excessive and damaging over-arousal. Anxiety thus serves a signal function, but also enables the organism to gain mastery of the source of stress through the development of an effective arousal modulating system. The importance of approaches such as this is that they do not automatically assume a spurious parallelism between psychological and physiological functioning, but provide a role for both in the healthy functioning of the organism.

One implication of a theory such as this is that constitutional differences may be very important indeed in the psychological development of young organisms, since it is in childhood that the systems for coping with physiological arousal are developed. It is possible, therefore, that a consideration of adverse environmental and constitutional factors can interact to produce an ineffective rousal modulating system, which has been claimed by Claridge (1967, op cit.), and Epstein, (1970) to underlie the psychological abnormalities present in schizophrenia. At a less dramatic level, such differences could easily be the cause of marked individual differences in personality in adult life.

* Of course I exclude Freud's theories from this criticism, but even the most ardent Freudian might question their physiological basis today.

It is not implausible to postulate psychophysiological stages of personality and emotional development, in the same way that Piaget has done for intellectual growth. This longitudinal perspective on the interrelationships between physiological and psychological measures, psyche and soma, is inevitably bound to be complex, but it is a necessary step in trying to comprehend the full complexity that Walt Whitman understood so well when he wrote -

"Of physiology from top to toe I sing
Not Physiognomy alone nor brain alone is worthy for the
Muse, I say the Form complete is worthier far ....."

One's self I sing.

Leaves of Grass, 1865.

EYSENCK PERSONALITY INVENTORY

by H. J. Eysenck and Sybil B. G. Eysenck

PERSONALITY QUESTIONNAIRE

FORM A

NAME..... AGE.....

OCCUPATION..... SEX.....

N= ☐ E= ☐ L= ☐

Instructions

Here are some questions regarding the way you behave, feel and act. After each question is a space for answering "YES" or "NO".

Try to decide whether "YES" or "NO" represents your usual way of acting or feeling. Then put a cross in the circle under the column headed "YES" or "NO". Work quickly, and don't spend too much time over any question; we want your first reaction, not a long-drawn out thought process. The whole questionnaire shouldn't take more than a few minutes. Be sure not to omit any questions.

Now turn the page over and go ahead. Work quickly, and remember to answer every question. There are no right or wrong answers, and this isn't a test of intelligence or ability, but simply a measure of the way you behave.

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E ○ N ○ L ○

FORM A

		YES	NO
1.	Do you often long for excitement?	○	○
2.	Do you often need understanding friends to cheer you up?	○	○
3.	Are you usually carefree?	○	○
4.	Do you find it very hard to take no for an answer?	○	○
5.	Do you stop and think things over before doing anything?	○	○
6.	If you say you will do something do you always keep your promise, no matter how inconvenient it might be to do so?	○	○
7.	Does your mood often go up and down?	○	○
8.	Do you generally do and say things quickly without stopping to think?	○	○
9.	Do you ever feel "just miserable" for no good reason?	○	○
10.	Would you do almost anything for a dare?	○	○
11.	Do you suddenly feel shy when you want to talk to an attractive stranger?	○	○
12.	Once in a while do you lose your temper and get angry?	○	○
13.	Do you often do things on the spur of the moment?	○	○
14.	Do you often worry about things you should not have done or said?	○	○
15.	Generally, do you prefer reading to meeting people?	○	○
16.	Are your feelings rather easily hurt?	○	○
17.	Do you like going out a lot?	○	○
18.	Do you occasionally have thoughts and ideas that you would not like other people to know about?	○	○
19.	Are you sometimes bubbling over with energy and sometimes very sluggish?	○	○
20.	Do you prefer to have few but special friends?	○	○
21.	Do you daydream a lot?	○	○
22.	When people shout at you, do you shout back?	○	○
23.	Are you often troubled about feelings of guilt?	○	○
24.	Are *all* your habits good and desirable ones?	○	○
25.	Can you usually let yourself go and enjoy yourself a lot at a gay party?	○	○
26.	Would you call yourself tense or "highly-strung"?	○	○
27.	Do other people think of you as being very lively?	○	○

		YES	NO
28.	After you have done something important, do you often come away feeling you could have done better?	○	○
29.	Are you mostly quiet when you are with other people?	○	○
30.	Do you sometimes gossip?	○	○
31.	Do ideas run through your head so that you cannot sleep?	○	○
32.	If there is something you want to know about, would you rather look it up in a book than talk to someone about it?	○	○
33.	Do you get palpitations or thumping in your heart?	○	○
34.	Do you like the kind of work that you need to pay close attention to?	○	○
35.	Do you get attacks of shaking or trembling?	○	○
36.	Would you always declare *everything* at the customs, even if you knew that you could never be found out?	○	○
37.	Do you hate being with a crowd who play jokes on one another?	○	○
38.	Are you an irritable person?	○	○
39.	Do you like doing things in which you have to act quickly?	○	○
40.	Do you worry about awful things that might happen?	○	○
41.	Are you slow and unhurried in the way you move?	○	○
42.	Have you ever been late for an appointment or work?	○	○
43.	Do you have many nightmares?	○	○
44.	Do you like talking to people so much that you never miss a chance of talking to a stranger?	○	○
45.	Are you troubled by aches and pains?	○	○
46.	Would you be very unhappy if you could not see lots of people most of the time?	○	○
47.	Would you call yourself a nervous person?	○	○
48.	Of all the people you know, are there some whom you definitely do not like?	○	○
49.	Would you say that you were fairly self-confident?	○	○
50.	Are you easily hurt when people find fault with you or your work?	○	○
51.	Do you find it hard to really enjoy yourself at a lively party?	○	○
52.	Are you troubled with feelings of inferiority?	○	○
53.	Can you easily get some life into a rather dull party?	○	○
54.	Do you sometimes talk about things you know nothing about?	○	○
55.	Do you worry about your health?	○	○
56.	Do you like playing pranks on others?	○	○
57.	Do you suffer from sleeplessness?	○	○

LEASE CHECK TO SEE THAT YOU HAVE ANSWERED *ALL* THE QUESTIONS

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FORM B
(1961 Edition)

WHAT TO DO: Inside this booklet are some questions to see what attitudes and interests you have. There are no "right" and "wrong" answers because everyone has the right to his own views. To be able to get the best advice from your results, you will want to answer them exactly and truly.

If a separate "Answer Sheet" has not been given to you, turn this booklet over and tear off the Answer Sheet on the back page.

Write your name and other particulars at the top of the Answer Sheet.

First, you should answer the four sample questions below so that you can see whether you need to ask anything before starting. Although you are to read the questions in this booklet, you must record your answers on the answer sheet (alongside the same number as in the booklet).

There are three possible answers to each question. Read the following examples and mark your answers at the top of your answer sheet where it says "Examples." Put a mark, x, in the left-hand box if your answer choice is the "a" answer, in the middle box if your answer choice is the "b" answer, and in the right-hand box if you choose the "c" answer.

EXAMPLES:

1. I like to watch team games. **(a) yes, (b) occasionally, (c) no.**
2. I prefer people who:
   (a) are reserved, (b) (are) in between, (c) make friends quickly.
3. Money cannot bring happiness. **(a) yes (true), (b) in between, (c) no (false).**
4. Woman is to child as cat is to: **(a) kitten, (b) dog, (c) boy.**

In the last example there *is* a right answer—kitten. But there are very few such reasoning items among the questions.

Ask *now* if anything is not clear. The examiner will tell you in a moment to turn the page and start.

When you answer, keep these four points in mind:

1. You are asked not to spend time pondering. **Give the first, natural answer as it comes to you.** Of course, the questions are too short to give you all the particulars you would sometimes like to have. For instance, the above question asks you about "team games" and you might be fonder of football than basketball. But you are to reply "for the average game," or to strike an average in situations of the kind stated. Give the best answer you can at a rate not slower than five or six a minute. You should finish in a little more than half an hour.
2. Try **not** to fall back on the middle, "uncertain" answers except when the answer at either end is really **impossible** for you—perhaps once every two or three questions.
3. Be sure not to skip anything, **but answer every question, somehow.** Some may not apply to you very well, but give your best guess. Some may seem personal; but remember that the answer sheets are kept confidential and cannot be scored without a special stencil key. Answers to particular questions are not inspected.
4. Answer as honestly as possible what is true of **you.** Do not merely mark what seems "the right thing to say" to impress the examiner.

DO NOT TURN PAGE UNTIL TOLD TO DO SO

1. I have the instructions for this test clearly in mind. **(a) yes, (b) uncertain, (c) no.**

2. I am ready to answer each question as truthfully as possible. **(a) yes, (b) in between, (c) no.**

3. For a vacation I would rather go to: **(a) a well-attended holiday town, (b) something in between (a) and (c), (c) a quiet cottage off the beaten track.**

4. When I'm in a small, cramped space (as on a crowded elevator), I have an uncomfortable feeling of being "shut in." **(a) never, (b) rarely, (c) occasionally.**

5. I find myself thinking over quite trivial troubles again and again and have to make a real effort to put them out of my mind. **(a) yes (true), (b) occasionally, (c) no (false).**

6. I like to be told how to do things instead of finding out for myself. **(a) yes, (b) uncertain, (c) no.**

7. My ideas appear to be: **(a) ahead of the times, (b) uncertain, (c) with the times.**

8. I am not much given to cracking jokes and telling amusing stories. **(a) true, (b) in between, (c) false.**

9. It is better to live to a good old age than to be worn out with patriotic service for one's community. **(a) true, (b) in between, (c) false.**

10. I have been active in organizing a club, team, or similar social group. **(a) yes, often, (b) occasionally, (c) never.**

11. I just can't help feeling sentimental. **(a) occasionally, (b) frequently, (c) often.**

12. I would rather read a book on: **(a) Great Religious Teachings, (b) uncertain, (c) Our National Political Organization.**

13. The topics on which I am "touchy" or easily annoyed are very few indeed. **(a) true, (b) uncertain, (c) false.**

14. The abilities and characteristics inherited from the parents are more important than many people are ready to admit. **(a) true, (b) in between, (c) false.**

15. I am always polite and diplomatic with unreasonable, unimaginative people and do not believe in showing up how narrow-minded they are. **(a) true, (b) in between, (c) false.**

16. Jokes about death are all right and normally in good taste. **(a) yes, (b) in between, (c) no.**

17. In physical and mental work I have to plan rest pauses, more than most people, if I am to keep up my best level of work. **(a) yes, (b) in between, (c) no.**

18. In the midst of social groups, I am nevertheless sometimes overcome by feelings of loneliness and worthlessness. **(a) yes, (b) in between, (c) no.**

19. I am brought almost to tears by having things go badly wrong. **(a) never, (b) occasionally, (c) often.**

20. I think divorce should be subject to fewer old-fashioned restrictions and be more a matter of mutual consent. **(a) yes, (b) in between, (c) no.**

21. For relaxation I prefer: **(a) sports or games, (b) uncertain, (c) debates and intellectual games.**

22. I find it easy to think out my own plans. **(a) not usually, (b) usually, (c) always.**

23. In traveling abroad I would rather go on a well-conducted tour than plan myself the places I wish to visit. **(a) yes, (b) uncertain, (c) no.**

24. In going places, eating, working, etc., I: **(a) seem to rush from one thing to another, (b) in between, (c) go in a deliberate, methodical way.**

25. I feel restless as if I want something but do not know what. **(a) very rarely, (b) occasionally, (c) often.**

(End of first column on answer sheet.)

26. In a factory, I would rather be in charge of: **(a) mechanical matters, (b) in between, (c) interviewing and hiring people.**

27. I would prefer to read a book on: **(a) travel in outer space, (b) uncertain,** (c) **education within the family.**

28. Which of the following words is not the same kind as the others? **(a) dog, (b) stone, (c) cow.**

29. If I had my life to live over again, I would: **(a) plan it differently, (b) uncertain, (c) want it much the same.**

30. In making decisions in my life and work, I was never troubled by lack of understanding on the part of my parents. **(a) true, (b) in between, (c) false.**

31. I have had accidents because I was deep in thought. **(a) hardly ever, (b) in between, (c) several times.**

32. If I had a gun in my hand that I knew was loaded, I would feel nervous until I unloaded it. **(a) yes, (b) in between, (c) no.**

33. I greatly like playing practical jokes with no malice in them. **(a) true, (b) in between, (c) false.**

34. People use up too much of their leisure in neighborly duties and helping with local affairs. **(a) yes, (b) uncertain, (c) no.**

35. Sometimes I feel that I do not do so well as I should socially, because I am unsure of myself. **(a) true, (b) in between, (c) false.**

36. In joining a new group, I seem to fit in immediately. **(a) yes, (b) uncertain, (c) no.**

37. The newspaper headline that would interest me more is: **(a) "Religious Leaders Discuss a Unified Creed," (b) uncertain, (c) "Improvements in Production and Marketing."**

38. I doubt the honesty of people who are more friendly than I would expect them to be. **(a) true, (b) in between, (c) false.**

39. I would prefer the life of: **(a) an artist or naturalist, (b) uncertain, (c) public accountant or insurance man.**

40. What this world needs is: **(a) more "idealists" with plans for a better world,** (b) **uncertain,** (c) **more steady and "solid" citizens.**

41. It embarrasses me to have servants waiting on me. **(a) yes, (b) in between,** (c) **no.**

42. I like a job that presents me with some subtle decisions rather than one with quick, routine answers. **(a) true, (b) uncertain, (c) false.**

43. I have a feeling that my friends do not need me so much as I need them. **(a) true, (b) uncertain, (c) false.**

44. I seldom get so taken up in a task that I forget where I put the necessary things. **(a) true, (b) uncertain, (c) false.**

45. With good salary, I could take the job of an attendant, looking after mental patients. **(a) yes, (b) in between, (c) no.**

46. In my newspaper, I like to see: **(a) debate on basic social issues in the modern world,** (b) **in between, (c) good coverage of all local news.**

47. I have been elected to: **(a) only a few offices, (b) several, (c) many offices.**

48. However difficult and unpleasant the obstacles, I always persevere and stick to my original intentions. **(a) yes, (b) in between, (c) no.**

49. My nerves get on edge, so that certain sounds, for example, a screechy hinge, are unbearable and give me the "shivers." **(a) often, (b) sometimes, (c) never.**

50. I often feel quite fatigued when I get up in the morning. **(a) yes, (b) in between, (c) no.**

(End of second column on answer sheet.)

51. With equal salary, I would prefer to be: **(a) a research chemist, (b) uncertain, (c) a hotel manager (or manageress).**

52. Going around selling things, or asking for funds to help a cause I believe in, is, for me: **(a) quite enjoyable, (b) in between, (c) an unpleasant job.**

53. Which one of the following three numbers does not belong with the others? **(a) 5, (b) 2, (c) 7.**

54. "Dog" is to "bone" as "cow" is to: **(a) milk, (b) grass, (c) salt.**

55. I would prefer to lead: **(a) the same kind of life I now lead, (b) uncertain, (c) a more sheltered life, with fewer difficulties to face.**

56. In a strange city, I would: **(a) walk wherever I liked, (b) uncertain, (c) avoid the parts of the town said to be dangerous.**

57. It is more important to: **(a) get along smoothly, (b) in between, (c) get your own ideas put into practice.**

58. I believe in: **(a) the motto "laugh and be merry" on most occasions, (b) in between, (c) being properly serious in everyday business.**

59. When given a set of rules, I follow them whenever personally convenient, rather than exactly to the letter. **(a) true, (b) uncertain, (c) false.**

60. In my social relations, I am sometimes troubled by a sense of inferiority, for which there is no real cause. **(a) yes, (b) in between, (c) no.**

61. When friends are in a lively conversation, I: **(a) prefer sometimes to be a shrewd listener, (b) in between, (c) get more remarks in than most people.**

62. I would rather: **(a) work with several people under me, (b) uncertain, (c) work with a committee.**

63. I practically never have to listen and take orders from people who are really stupid. **(a) true, (b) uncertain, (c) false.**

64. No one would really like to see me in trouble. **(a) true, (b) uncertain, (c) false.**

65. It is more important for a man to be concerned about: **(a) the basic meaning of life, (b) uncertain, (c) making a good income for his family.**

66. Being long indoors, away from the fresh outdoors, makes me feel stale. **(a) always, (b) sometimes, (c) hardly ever.**

67. I am sometimes so very happy that I get afraid my happiness cannot last. **(a) true, (b) in between, (c) false.**

68. My spirits generally stay high, no matter how much trouble I meet. **(a) true, (b) in between, (c) false.**

69. It generally makes me unhappy when I have to move all my belongings to a new place. **(a) true, (b) in between, (c) false.**

70. I would enjoy technical work on the stock exchange, working out trends, etc. **(a) yes, (b) in between, (c) no.**

71. My friends probably think it is hard to get to know me really well. **(a) yes, (b) in between, (c) no.**

72. I solve a problem better by: **(a) studying it alone, (b) in between, (c) discussing it with others.**

73. When quick decisions must be made, I: **(a) rely on calm, logical, and objective reasoning, (b) in between, (c) become tense and excitable, unable to think clearly.**

74. I sometimes find quite useless thoughts and memories straying through my mind. **(a) yes, (b) in between, (c) no.**

75. I never find myself so annoyed in discussions that I cannot control my voice. **(a) true, (b) uncertain, (c) false.**

(End of third column on answer sheet.)

76. When traveling, I would rather look at the scenery than talk to people. **(a) true, (b) uncertain, (c) false.**

77. Is "lose" a better opposite to "reveal" than "hide"? **(a) yes, (b) uncertain, (c) no.**

78. "Black" is to "gray" as "pain" is to: **(a) wound, (b) discomfort, (c) illness.**

79. I find it hard to "take 'no' for an answer," even when I know I ask the impossible. **(a) true, (b) in between, (c) false.**

80. I am often hurt more by the way people say things than by what they say. **(a) true, (b) in between, (c) false.**

81. I dislike people who are too self confident and act as if they are superior to the general run of humanity. **(a) true, (b) in between, (c) false.**

82. I find it difficult, the moment I stop work, to put work out of my mind and relax. **(a) true, (b) in between, (c) false.**

83. I like being amidst a great deal of excitement and bustle. **(a) yes, (b) in between, (c) no.**

84. At work it is really more important to be popular with the right people than to do a first-rate job. **(a) true, (b) in between, (c) false.**

85. If people in the street, or standing in a store, watch me, I feel slightly embarrassed. **(a) yes, (b) in between, (c) no.**

86. My ideas cannot always be easily put into words, so I do not cut into a conversation as readily as most people do. **(a) true, (b) in between, (c) false.**

87. I believe in complaining to the waiter or manager if I am served bad food in a restaurant. **(a) yes, (b) in between, (c) no.**

88. It is mainly the fear of being caught that keeps most people from dishonest or criminal acts. **(a) yes, (b) in between, (c) no.**

89. In demanding and enforcing obedience, my parents (or guardians) were always very mild. **(a) true (b) uncertain, (c) false.**

90. I wish society would demand: (a) **stricter observance of the Sabbath, (b) uncertain, (c) a greater freedom in regard to divorce.**

91. I get unusual ideas about all sorts of things—too many to put into practice. **(a) yes, (b) in between, (c) no.**

92. I enjoy routine, constructive work, using a good piece of machinery or apparatus. **(a) yes, (b) in between, (c) no.**

93. On top of a high building or in a deep tunnel, I never feel nervous. **(a) true, (b) uncertain, (c) false.**

94. I am troubled by feelings of guilt or remorse over quite small matters. **(a) yes, often, (b) sometimes, (c) no.**

95. I sometimes make foolish remarks in fun, just to surprise people and see what they will say. **(a) yes, (b) in between, (c) no.**

96. We should direct our lives more by: **(a) the standards of our group, (b) in between, (c) our own individual reasoning.**

97. Many people talk over their problems and ask advice of me when they need someone to talk to. **(a) yes, (b) in between, (c) no.**

98. One should be careful about mixing with all kinds of strangers, for there are dangers of infection and so on. **(a) yes, (b) uncertain, (c) no.**

99. In some moods I get easily put off my work by distractions and daydreams. **(a) yes, (b) in between, (c) no.**

100. I do not get immediate likes and dislikes for people I have just met. **(a) true, (b) uncertain, (c) false.**

(End of fourth column on answer sheet.)

101. I would prefer to be: **(a) business office manager, (b) uncertain, (c) an architect.**

102. "April" is to "March" as "Tuesday" is to: **(a) Wednesday, (b) Friday, (c) Monday.**

103. Which of the following words does not belong with the others? **(a) wise, (b) lovely, (c) kind.**

104. I cross the street to avoid meeting people I don't feel like seeing. **(a) seldom, (b) occasionally, (c) sometimes.**

105. I can always change old habits without difficulty and without slipping back. **(a) yes, (b) in between, (c) no.**

106. If I disagreed with a class teacher on his views, I would usually: **(a) keep my opinion to myself, (b) uncertain, (c) tell him in class that my opinion differs.**

107. I avoid any embarrassing sexual topic in talking with members of the opposite sex. **(a) yes, (b) in between, (c) no.**

108. I am not really successful in dealing with people. **(a) true, (b) uncertain, (c) false.**

109. I enjoy giving my best time and energy to: **(a) my home and the real needs of my friends, (b) in between, (c) social activities and personal hobbies.**

110. When I wish to impress people favorably with my personality, I: **(a) nearly always succeed, (b) sometimes succeed, (c) am generally uncertain of success.**

111. I prefer to have: **(a) a large circle of acquaintances, (b) uncertain, (c) just a few, well-tried friends.**

112. I would rather be a philosopher than a mechanical engineer. **(a) true, (b) uncertain, (c) false.**

113. I believe anyone will tell a lie to keep out of trouble. **(a) true, (b) uncertain, (c) false.**

114. I enjoy planning carefully to influence my associates so that they will help me in achieving my goals. **(a) true, (b) in between, (c) false.**

115. I have occasionally had a brief touch of faintness, dizziness, or light-headedness for no apparent reason. **(a) yes, (b) uncertain, (c) no.**

116. My friends think I am slightly absent-minded and impractical. **(a) yes, (b) uncertain, (c) no.**

117. I would rather vote for: **(a) operations to stop mental defectives from having children, (b) uncertain, (c) the death penalty for murder.**

118. When I seem unable to finish a task as promised, I: **(a) push to finish it, even to the point of hurting myself and others, (b) try just a bit harder, (c) never worry.**

119. I occasionally have periods of feeling depressed, miserable, and in low spirits for no sufficient reason. **(a) yes, (b) in between, (c) no.**

120. In my work more troubles arise from people who: **(a) are constantly changing methods that are already O.K., (b) uncertain, (c) refuse to employ up-to-date methods.**

121. A person whose ambitions hurt and damage a close friend may yet be considered an ordinary, decent citizen. **(a) yes, (b) in between, (c) no.**

122. When looking for a place in a strange city, I would: **(a) just ask people where places are, (b) in between, (c) take a map with me.**

123. I sometimes stir up friends to go out when they say they really want to stay home. **(a) yes, (b) uncertain, (c) no.**

124. When going to bed, I: **(a) have difficulty falling asleep, (b) in between, (c) drop off to sleep quickly and normally.**

125. If someone annoys me, I: **(a) can keep it to myself, (b) in between, (c) must speak to someone else to "let off steam."**

(End of fifth column on answer sheet.)

126. I would rather be an insurance salesman than a farmer. **(a) yes, (b) in between, (c) no.**

127. "Statue" is to "shape" as "song" is to: **(a) beauty, (b) notes, (c) tune.**

128. Which of the following words does not belong with the others? **(a) hum, (b) speak, (c) whistle.**

129. I feel that modern life has too many annoying frustrations and restrictions. **(a) yes, (b) in between, (c) no.**

130. I am much more fortunate than most in being able to do the things I like. **(a) yes, (b) uncertain, (c) no.**

131. When I need immediately the use of something belonging to a friend but he is out, I think it is all right to borrow it without his permission. **(a) yes, (b) in between, (c) no.**

132. Nearly always I have a craving for more excitement. **(a) true, (b) in between, (c) false.**

133. I would prefer to be: **(a) an actor, (b) uncertain, (c) a house builder.**

134. I find it desirable to make plans to avoid waste of time between jobs. **(a) yes, (b) in between, (c) no.**

135. In a group I am usually: **(a) well in touch with all that goes on around me, (b) in between, (c) wrapped up in my own thoughts or immediate business.**

136. I enjoy getting into conversation, and I rarely let a chance go by to speak to a stranger. **(a) true, (b) in between, (c) false.**

137. I strongly enjoy the racy and slap-stick humor of the usual television vaudeville show. **(a) yes, (b) in between, (c) no.**

138. I enjoy daydreaming. **(a) yes, (b) uncertain, (c) no.**

139. International affairs in the next twenty years are likely to get better rather than worse. **(a) true, (b) uncertain, (c) false.**

140. I am less interested in being practically successful than in seeking artistic and spiritual truths. **(a) true, (b) in between, (c) false.**

141. As a child I enjoyed better: **(a) stories of battle and conquests, (b) uncertain, (c) imaginative fairy tales.**

142. I would like to work as a probation officer with criminals on parole. **(a) yes, (b) in between, (c) no.**

143. I find myself upset rather than helped by the kind of criticism that many people offer one. **(a) often, (b) occasionally, (c) never.**

144. Inconsiderate acts or remarks by my neighbors do not make me touchy and unhappy. **(a) true, (b) uncertain, (c) false.**

145. Society should be guided more by logical thinking and less by sentimental, traditional beliefs. **(a) yes, (b) in between, (c) no.**

146. When pushed and overworked, I suffer from indigestion or constipation. **(a) occasionally, (b) hardly ever, (c) never.**

147. I tend to get over-excited and "rattled" in upsetting situations. **(a) yes, (b) in between, (c) no.**

148. I make a point of not being absent-minded, or forgetful of details. **(a) yes, (b) in between, (c) no.**

149. A near-accident, or even a lively argument, sometimes leaves me shaky and exhausted, so that I cannot settle down to what I was doing. **(a) true, (b) in between, (c) false.**

150. I never take medicine on my own without waiting for a doctor to order it. **(a) true, (b) in between, (c) false.**

(End of sixth column on answer sheet.)

151. For a pleasant hobby I would rather belong to: **(a) a photographic club, (b) uncertain, (c) a debating society.**

152. "Combine" is to "mix" as "team" is to: **(a) crowd, (b) army, (c) football.**

153. "Clock" is to "time" as "tailor" is to: **(a) suit, (b) scissors, (c) cloth.**

154. I have difficulty in following what some people are trying to say because of their odd use of common words. **(a) yes, (b) in between, (c) no.**

155. I have on occasion torn down a public notice forbidding me what I feel I had a perfect right to do. **(a) yes, (b) in between, (c) no.**

156. People have sometimes called me a proud, "stuck-up" individual. **(a) yes, (b) in between, (c) no.**

157. I would prefer the life of a master printer to that of an advertising man and promoter. **(a) true, (b) uncertain, (c) false.**

158. I can tell a fantastic lie with a perfectly straight face. **(a) never, (b) with some difficulty, (c) easily, if it is fun to do so.**

159. When I do something, my main concern is that: **(a) it is really what I want to do, (b) uncertain, (c) no bad consequences will follow for my associates.**

160. When people need my decisions, I don't keep them waiting even an hour more than necessary. **(a) true, (b) uncertain, (c) false.**

161. Starting up conversations with strangers: **(a) is rather difficult for me, (b) in between, (c) never gives me the least trouble.**

162. Upsetting the dignity of teachers, judges, and "cultured" people always amuses me. **(a) yes, (b) in between, (c) no.**

163. I think I am more sensitive than most people to the artistic quality of my surroundings. **(a) yes, (b) uncertain, (c) no.**

164. I get irritated by people who adopt morally superior attitudes. **(a) yes, (b) in between, (c) no.**

165. I would rather spend time enjoying: **(a) a game of cards with a congenial group, (b) uncertain, (c) the beautiful things in an art gallery.**

166. I can take a position in an argument, just to give all sides a chance, even when I do not believe in it deeply myself. **(a) yes, (b) in between, (c) no.**

167. I enjoy talking more with polished, sophisticated people than with outspoken, down-to-earth individuals. **(a) yes, (b) in between, (c) no.**

168. Even when the blame can be put on others, most people don't mind admitting their guilt. **(a) true, (b) uncertain, (c) false.**

169. When I reason with someone on a difference of social views, I like: **(a) to find out basically what our difference means, (b) uncertain, (c) to reach a practical way of working, satisfactory to both.**

170. In most of the difficulties I meet in work and society, my own mistakes are largely to blame. **(a) true, (b) in between, (c) false.**

171. I get as many ideas from reading a book myself as from discussing its topics with others. **(a) yes, (b) in between, (c) no.**

172. I would rather take the gamble of a job with possibly large but uneven earnings, than one with a steady, small salary. **(a) yes, (b) uncertain, (c) no.**

173. If people think poorly of me, I can still go on serenely in my own mind. **(a) yes, (b) in between, (c) no.**

174. Newspaper accounts of everyday dangers and accidents: **(a) hold my attention, (b) in between, (c) make rather dull, trivial reading.**

175. I would rather live in an up-and-coming town than in a quiet country village. **(a) true, (b) uncertain, (c) false.**

(End of seventh column on answer sheet.)

176. I would enjoy better: **(a) being in charge of children's games, (b) uncertain, (c) helping a watchmaker.**

177. "Justice" is to "laws" as "idea" is to: **(a) words, (b) feelings, (c) judges.**

178. Which of the following words does not belong with the others? **(a) second, (b) once, (c) alone.**

179. Changes in weather are generally powerless to affect my performance or mood. **(a) true, (b) in between, (c) false.**

180. Government lawyers are mainly interested in: (a) **making convictions, regardless of the person, (b) uncertain, (c) protecting the innocent.**

181. My speaking voice is: **(a) strong, (b) in between, (c) soft.**

182. I like acting on impulses of the moment, even if they land me in later difficulties. **(a) yes, (b) in between, (c) no.**

183. I am well described as a happy-go-lucky, nonchalant person. **(a) yes, (b) in between, (c) no.**

184. I never admire even a really successful criminal or confidence man. **(a) true, (b) in between, (c) false.**

185. I always check very carefully the condition in which borrowed property is returned, to me or by me to others. **(a) yes, (b) in between, (c) no.**

186. In social groups I am bothered by self-conscious shyness. (a) **never, (b) sometimes, (c) often.**

187. I am sure there are no questions that I have not answered properly. **(a) yes, (b) uncertain, (c) no.**

(Do not tear off this sheet unless told to do so.)

'PAT

ANSWER SHEET: THE 16 P. F. TEST, FORM B

NAME _____ SEX _____ AGE _____ DATE _____ ★

First Middle Last (Write M or F) (Nearest Year)

EXAMPLES: 1 a b c 2 a b c 3 a b c 4 a b c

RAW SCORE

A B C E F G H I L M N O Q_1 Q_2 Q_3 Q_4

BE SURE YOUR MARKS ARE HEAVY. ERASE COMPLETELY ANY ANSWER YOU WISH TO CHANGE.

END OF TEST

THE HOSTILITY AND DIRECTION OF HOSTILITY QUESTIONNAIRE

NAME:

AGE:

SEX:

OCCUPATION:

DATE:

Instructions: Please fill in this form by putting a circle round the "True" or the "False" following each statement. If you find it difficult to decide, ask yourself whether you think the statement is on the whole true or false and put a circle round the appropriate word.

Remember to answer each statement

1.	Most people make friends because friends are likely to be useful to them.	True	False
2.	I do not blame a person for taking advantage of someone who lays himself open to it.	True	False
3.	I usually expect to succeed in things I do.	True	False
4.	I have no enemies who really wish to harm me.	True	False
5.	I wish I could get over worrying about things I have said that may have injured other people's feelings.	True	False
6.	I think nearly anyone would tell a lie to keep out of trouble.	True	False
7.	I don't blame anyone for trying to grab everything he can get in this world.	True	False
8.	My hardest battles are with myself.	True	False
9.	I know who, apart from myself, is responsible for most of my troubles.	True	False
10.	Some people are so bossy that I feel like doing the opposite of what they request, even though I know they are right.	True	False

11.	Some of my family have habits that bother and annoy me very much.	True	False
12.	I believe my sins are unpardonable.	True	False
13.	I have very few quarrels with members of my family.	True	False
14.	I have often lost out on things because I couldn't make up my mind soon enough.	True	False
15.	I can easily make other people afraid of me, and sometimes do for the fun of it.	True	False
16.	I believe I am a condemned person.	True	False
17.	In school I was sometimes sent to the principal for misbehaving.	True	False
18.	I have at times stood in the way of people who were trying to do something, not because it amounted to much but because of the principle of the thing.	True	False
19.	Most people are honest chiefly through fear of being caught.	True	False
20.	Sometimes I enjoy hurting persons I love.	True	False
21.	I have not lived the right kind of life.	True	False
22.	Sometimes I feel as if I must injure either myself or someone else.	True	False
23.	I seem to be about as capable and clever as most others around me.	True	False
24.	I sometimes tease animals.	True	False
25.	I get angry sometimes.	True	False
26.	I am entirely self-confident.	True	False
27.	Often I can't understand why I have been so cross and grouchy.	True	False
28.	I shrink from facing a crisis or difficulty.	True	False

29. I think most people would lie to get ahead. True False

30. I have sometimes felt that difficulties were piling up so high that I could not overcome the. True False

31. If people had not had it in for me I would have been much more successful. True False

32. I have often found people jealous of my good ideas, just because they had not thought of them first. True False

33. Much of the time I feel as if I have done something wrong or evil. True False

34. I have several times given up doing a thing because I thought too little of my ability. True False

35. Someone has it in for me.

36. When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing. True False

37. I am sure I get a raw deal from life. True False

38. I believe I am being followed. True False

39. At times I have a strong urge to do something harmful or shocking. True False

40. I am easily downed in an argument. True False

41. It is safer to trust nobody. True False

42. I easily become impatient with people. True False

43. At times I think I am no good at all. True False

44. I commonly wonder what hidden reason another person may have for doing something nice for me. True False

45. I get angry easily and then get over it soon. True False

46.	At times I feel like smashing things.	True	False
47.	I believe I am being plotted against.	True	False
48.	I certainly feel useless at times.	True	False
49.	At times I feel like picking a fist fight with someone.	True	False
50.	Someone has been trying to rob me.	True	False
51.	I am certainly lacking in self-confidence.	True	False

Please check to see that you have given answers for every statement.

THE TAYLOR MANIFEST ANXIETY SCALE

SELF-RATING QUESTIONNAIRE

Name: _____ Trial No.: _____

Date: _____ Rating No.: _____

Total Score: _____

Instructions: Please fill in this form by putting a circle around the "True" or "False" following each statement. If you find it difficult to decide, ask yourself whether you think the statement is on the whole true or false and put a circle round the appropriate word.

REMEMBER TO ANSWER EACH STATEMENT

1. My sleep is fitful and disturbed . . . . . True False
2. I have had periods in which I lost sleep over worry . . . . . True False
3. I have very few fears compared with my friends . . True False
4. I believe I am no more nervous than most others . . True False
5. I have nightmares every few nights . . . . . True False
6. I have had a great deal of stomach trouble . . . . True False
7. I frequently notice my hand shake when I try to do something . . . . . True False
8. I suffer from attacks of diarrhoea . . . . . True False
9. I worry over money and business . . . . . True False
10. I am troubled by attacks of nausea . . . . . True False
11. I am often afraid that I am going to blush . . . . True False
12. I feel hungry almost all the time . . . . . True False

13. I am entirely self-confident . . . . . True False

14. I do not tire quickly . . . . . True False

15. It makes me nervous to have to wait . . . . . True False

16. Sometimes I become so excited that I find it hard to get to sleep . . . . . True False

17. I am usually calm and not easily upset . . . . . True False

18. I have periods of such great restlessness that I cannot sit long in a chair . . . . . True False

19. I am happy most of the time .... True False

20. I find it hard to keep my mind on a task or job . True False

21. I feel anxiety about something or someone almost all the time . . . . . True False

22. I shrink from facing a crisis or difficulty . . . True False

23. I wish I could be as happy as others seem to be . True False

24. I frequently find myself worrying about something True False

25. I certainly feel useless at times . . . . . True False

26. I sometimes feel that I am about to go to pieces . True False

27. I sweat very easily even on cool days . . . . . True False

28. Life is a strain for me much of the time . . . . . True False

29. I worry over possible misfortune . . . . . True False

30. I am unusually self-conscious . . . . . True False

31. I hardly ever notice my heart pounding and I am seldom short of breath . . . . . True False

32. I cry easily . . . . . True False

33. I have been afraid of things or people that I know could not hurt me . . . . . True False

34. I am inclined to take things hard . . . . . True False

35. I have very few headaches . . . . . True False

36. I must admit that I have at times been worried beyond reason over something that really did not matter . . . . . True False

37. I cannot keep my mind on one thing . . . . . True False

38. I am easily embarrassed . . . . . True False

39. At times I think I am no good at all . . . . . True False

40. I am a highly strung person . . . . . True False

41. Sometimes when embarrased, I break out in a sweat which annoys me greatly . . . . . True False

42. I blush no more often than others . . . . . True False

43. I am more sensitive than most other people . . . True False

44. I practically never blush . . . . . True False

45. I have sometimes felt that difficulties were piling up so high that I could not overcome them . . . . . True False

46. I work under a great deal of tension . . . . . True False

47. My hands and feet are usuallly warm enough . . . . True False

48. I dream frequently about things that are best kept to myself . . . . . True False

49. I lack self-confidence . . . . . True False

50. I am very seldom troubled by constipation . . . . True False

APPENDIX 2

LISTING OF COMPUTER PROGRAMS

1: The regression analysis program

```
 %BEGIN
%REAL SLONG
%REALARRAY  A,C,D(1:52)
%INTEGER  %ARRAY  Z(1:50)
%INTEGER   I,J,K,N,M
%REAL    SUMX, SUMY, SQX, SQY, XY
%ROUTINE %SPEC REGRESSION (%REALARRAYNAME A,B,%INTEGERNAME M,N)
%ROUTINE  %SPEC  READNAME (%INTEGER  %ARRAY  %NAME  B)
%ROUTINE  %SPEC  PRINTNAME (%INTEGER  %ARRAY  %NAME  A)
1:READNAME(Z)
%CYCLE  I = 1,1,52
      READ(A(I)); D(I) = -1
-> 99 %IF  A(I)<0
D(I) = 0 %IF  12<=I<=31 %AND  A(I) =0
D(I-21) = 0 %IF   33<=I<=52 %AND  A(I) = 0
A(I) = 0.43429*LOG (1000/A(I)) %IF  A(I)#0
%REPEAT
%CYCLE  J = 0,1,7
K = 0
C(J+ 1) = 0
%CYCLE  I = 1,1,4
C(J + 1) = C(J + 1) + A(4*J + I)
K = K + 1 %IF  A(4*J + I) # 0
%REPEAT
K = 1 %IF  K =0
C(J + 1) = C(J + 1)/K
%REPEAT
%CYCLE   I = 12,1,31
D(I) = 0.43429*LOG(I - 11) %UNLESS  D(I) = 0 %OR  D(I+ 21) = 0
C(I) = 0 %IF  A(I) = 0 %OR  A(I +1 ) = 0
C(I) = A(I +21) - A(I)
%REPEAT
```

```
NEWPAGE
PRINTNAME(Z)
NEWLINES(2)
%PRINTTEXT'   LOG  CONDUCTANCES AND CHANGES'
NEWLINE
%CYCLE  I = 1,1,32
PRINT(A(I) ,1,4)
SPACES(3)
SPACES(7) %UNLESS  12<=I<=31
PRINT(A(I + 21), 1,4) %IF  12<=I<=31
SPACES(3)
PRINT(C(I),1,4) %IF   12<=I<=31
SPACES(7) %UNLESS  12<=I<=31
SPACES(3)
J = INT PT(I/4)
PRINT(C(J), 1,4) %IF  4*J = I
NEWLINE
%REPEAT
I = 13
N = 31
3: -> 6 %IF  I >= N + 1
->4 %IF  D(I) #0
J = I
5: C(J) = C(J+1) %UNLESS  J = N
D(J) = D(J+1) %UNLESS  J = N
J = J +1
->5 %UNLESS  J = N + 1
N = N - 1
-> 3
4: I = I + 1
-> 3
6: J = N
N = N - 1 %IF   C(N) = C %AND  C(N - 1) = 0 %AND  C(N -2) = 0 %AND  C%C
(N - 3) = 0
-> 6 %IF  J # N
M = 13
REGRESSION ( D,C,M,N)
-> 1
```

```
%ROUTINE  REGRESSION (%REALARRAY  %NAME  A,B, %INTEGERNAME  M,N)
%INTEGER  I
%REAL  RG,DV
SUMX = 0
SUMY = 0
SQX = 0
SQY = 0
XY = 0
%CYCLE   I = M,1,N
SUMX = SUMX + A(I)
SUMY = SUMY + B(I)
SQX = SQX + A(I)*A(I)
SQY = SQY + B(I)*B(I)
XY = XY + A(I)*B(I)
%REPEAT
NEWLINES(3)
%PRINTTEXT'LARGE X SQUARE'
SPACES(11)
PRINT(SQX,2,8)
%PRINTTEXT'
LARGE Y SQUARE'
SPACES(11)
PRINT(SQY,2,8)
%PRINTTEXT'
LARGE XY'
SPACES(17)
PRINT(XY,2,8)
NEWLINE
SQX = SQX - (SUMX**2/(N+1-M))
SQY = SQY - (SUMY**2/(N+1-M))
XY = XY - (SUMX*SUMY/(N+1-M))
%PRINTTEXT'REGRESSION  COEFFICIENT  '
PRINT (XY/SQX,2,8)
NEWLINE
%PRINTTEXT'CONSTANT  '
SPACES(15)
PRINT((SUMY/(N+1-M))-(XY/SQX)*(SUMX/(N+1-M)),2,8)
NEWLINE
%PRINTTEXT'SUM OF X SQUARE  '
SPACES(8)
PRINT(SQX,2,8)
%PRINTTEXT'
SUM OF Y SQUARE  '
SPACES(8)
PRINT(SQY,2,8)
NEWLINE
%PRINTTEXT'SUM OF XY    '
SPACES(12)
PRINT(XY,2,8)
NEWLINE
%PRINTTEXT'SUM OF X   '
```

```
SPACES(14)
PRINT(SUMX,2,8)
NEWLINE
%PRINTTEXT'SUM OF Y    '
SPACES(14)
PRINT(SUMY,2,8)
NEWLINE
%PRINTTEXT'NUMBER OF READINGS =    '
PRINT(N + 1 - M,3,0)
%PRINTTEXT'
F (1,'
PRINT(N - M -1,2,0)
%PRINTTEXT' ) ='
RG = XY*XY/SQX
DV = (SQY - RG)/(N - M - 1)
PRINT (RG/DV,2,8)
NEWLINE
%END
%ROUTINE  READNAME (%INTEGERARRAYNAME  B)
%INTEGER  I,J
I = 1
1: READ SYMBOL (J)
-> 1 %UNLESS  J = '('
2: READ SYMBOL(B(I))
%RETURN  %IF  B(I) = '?'  %OR  B(I) = ')'
->3 %IF  I > 50
I = I + 1
->2
3: %PRINTTEXT'FAULTY DATA TITLE'
%STOP
%END
%ROUTINE  PRINTNAME (%INTEGERARRAYNAME  A)
%INTEGER  I
NEWLINE
I = 1
1:%RETURN  %IF  A(I) = '?'  %OR A(I) = ')'
PRINT SYMBOL(A(I))
I = I + 1
-> 1
%END
99: %END  %OF  %PROGRAM
```

2: The READDATA program

```
%BEGIN
%SHORTINTEGERARRAY  FS(1:100,1:5)
      %STRING(6)%ARRAY  NAME(1:140)
%BYTEINTEGERARRAY  BB(1:50)
%BYTEINTEGERARRAY FORMAT(1:100)
%STRING(8) %ARRAY  DT(1:100)
      %STRING(15) %ARRAY  ML(1:3)
%STRING(1) COND
%STRING(72) LN
%INTEGER DISC,J,K
%SHORTINTEGER  TM,BLOCKS,FILEND
%BYTEINTEGER  NB,MODE,TR,SUM,SETNO,MCRE,ISTREAM
%ROUTINESPEC SQUASH(%STRINGNAME  S)
%ROUTINESPEC READLINE(%STRINGNAME LN,%BYTEINTEGER TR)
%REALFNSPEC S TO R(%STRINGNAME S,COND)
%INTEGERFNSPEC SECTIME(%REAL U)
%EXTERNALSTRINGFNSPEC  FROMSTRING(%STRINGNAME S,%INTEGER I,J)
%EXTERNALROUTINESPEC  READSTRING(%STRINGNAME  S)
      %ROUTINESPEC PACKUP
%EXTERNALSTRINGFNSPEC  TOSTRING(%INTEGER N)
     %ROUTINESPEC DECOMP(%STRING(80) DOP,%STRINGARRAYNAME WD, %C
%BYTEINTEGERNAME DE)
%EXTERNALSTRINGFNSPEC  DATE
%EXTERNALROUTINESPEC OPENDA(%INTEGER CHANNEL)
%EXTERNALROUTINESPEC CLOSEDA(%INTEGER CHANNEL)
%EXTERNALROUTINESPEC WRITEDA(%INTEGER CHANNEL,%INTEGERNAME SECT %C
,%NAME BEGIN,END)
%EXTERNALROUTINESPEC  READ DA(%INTEGER CHANN, %INTEGERNAME SECT %C
%NAME  BEG, END)
%EXTERNALROUTINESPEC READITEM(%STRINGM"NAME  S)
      %EXTERNALINTEGERFNSPEC CHARNO(%STRINGNAME S,%INTEGER  N)
      %EXTERNALINTEGERFNSPEC LENGTH(%STRINGNAME S)
      %EXTERNALSTRINGFNSPEC  NEXT ITEM
      %BYTEINTEGERMAPSPEC CV"CC(%INTEGER V)
%BYTEINTEGERMAP SCANLENGTH(%INTEGER J)
%RESULT = ADDR(FS(J,4)); %END
%BYTEINTEGERMAP  ISI(%INTEGER J)
%RESULT =ADDR(FS(J,4))+1 ; %END
%ROUTINE TITLE(%INTEGER K,L)
%IF L = 2 %THEN %PRINTTEXT'STRING TO REAL CONVERSION FAILED'
%IF   L=4 %THEN  %PRINTTEXT' DECLARED  ARRAY BOUNDS EXCEEDED'
%IF L = 5 %THEN %PRINTTEXT'WRONG NO OF SYLLABLES IN LINE '
%END
%FAULT  18 ->F9
%FAULT 9 ->F9
```

```
MORE=0
1: OPENDA(12)
DISC=201;READDA(12,DISC,FS(1,1),FS(100,5))
DISC=202;READDA(12,DISC,BYTEINTEGER(ADDR(NAME(1))), %C
BYTEINTEGER("(ADDR(NAME(100))+6))
DISC=203
READDA(12,DISC,BYTEINTEGER(ADDR(DT(1))),BYTEINTEGER(ADDR(DT(100)) %C
+8))
A1:SELECTINPUT(94)
TR=1
      READLINE(LN,TR);->F1 %UNLESS LN='B' %I"OR LN = 'BEGIN'
2:READLINE(LN,TR);DECOMP(LN,ML,NB)
->FREE %IF ML(1)='*FREEBLOCK'
->DEL %IF ML(1)='*DELETE'
-> GO %IF ML(1) ='GO'
->2 %UNLESS ML(1) = 'FILENAME'
25:NAME(100)=ML(2);SUM=1
%CYCLE J = 1,1,99; %IF NAME(J) =NAME(100) %THENSTART
PRINTSTRING('
*********NAME'.NAME(100).' USED TWICE'); ->22; %FINISH ; %REPEAT
3:   READLINE(LN,TR);DECOMP(LN,ML,NB)
%IF ML(1)= 'LENGTH' %THEN %START
SCANLENGTH(100)=INT(STOR(ML(2),COND)); ->F2 %IF COND='F'
SUM = SUM+1; ->3 ; %FINISH
%IF ML(1)='TIME' %THEN %START
TM = SECTIME(INT(STOR(ML(2),COND))); ->F2 %IF COND='F'
SUM = SUM +1 ; ->3; %FINISH
%IF ML(1)= 'MODE' %THEN %START
FORMAT(100)=0
FORMAT(100)=1 %IF ML(2)='EMS'
FORMAT(100)=2 %IF ML(2)='ETS'
FORMAT(100)=3 %IF ML(2)='VFAST'
FORMAT(J)=FORMAT(100)
->22 %IF FORMAT(100)=0;SUM=SUM+1;->3;%FINISH
%IF ML(1) = 'ISI' %THEN %START
ISI(100) = INT(STOR(ML(2),COND)); ->F2 %IF COND='F'
SUM = SUM + 1; ->3; %FINISH
      %IF  ML(1)= 'END' %THEN  ->ALLOCATE
%PRINTTEXT'
**********UNIDENTIFIABLE COMMAND'; PRINTSTRING(LN); ->2
ALLOCATE:
 %IF SUM <5 %THEN %START
%PRINTTEXT'
**********NOT ENOUGH PARAMETERS FOR FILENAME';PRINTSTRING(NAME(100))
->2
%FINISH
FS(100,1)=INT((TM*SCANLENGTH(100))/ISI(100))+SCANLENGTH(100)
FS(100,2) = INTPT((FS(100,1)-1)/512) + 1
BLOCKS = FS(100)",2)
%CYCLE J = 1,1,99
%IF FS(J,3)=0 %THEN ->5
%REPEAT; %PRINTTEXT'
**********NO SPACE IN CATALOGUE FOR ANY MORE DATA SETS '; ->22
5:  %CYCLE K = 1,1,4
FS(J,K) <- FS(100,K)
%REPEAT;  FS(J,3)= 1
```

```
NAME(J) = NAME(100);NAME(100)=''
FORMAT(J) = FORMAT(100); FORMAT(100) = ''
DT(J) =DATE
NB=1; %PRINTTEXT'
LOGICAL FILE NO. IS';WRITE(J,2);PRINTSTRING(' FOR FILENAME  '.NAME(J))
%PRINTTEXT'  DATA STORED IN BLOCKS '
%CYCLE K=1,1,200; %IF OCC(K)=0  %THENSTART
OCC(K) =J; BLOCKS =BLOCKS-1
WRITE(K,4); ->6 %IF BLOCKS = 0; %FINISH
%REPEAT; ->F3
6:
SETNO=SETNO + 1; NEWLINE; ->2
F3: %PRINTTEXT '
**********NO MORE BLOCKS FREE FOR DATA STORAGE '; ->22
F4:%PRINTTEXT '
**********TOO MANY DATA SETS IN THIS RUN ********'
->22
FREE:K=INT(STOR(ML(2),COND));->2 %IF COND#'I' %OR K<1 %OR K>200
OCC(K)=0;->2
DEL: ->2 %IF ML(2) = 'DUMMY'
%CYCLE J = 1,1,100
->20 %IF NAME(J) = ML(2); %REPEAT; NEWLINE
PRINTSTRING('**********DATA SET WITH NAME'.ML(2).'CANNOT BE FOUND')
->2
20: NAME(J) = ''; FS(J,3)=0; ISI(J)=0;SCANLENGTH(J)=0
NEWLINE; PRINTSTRING('DATA SET NAMED '. ML(2).' DELETED FROM BLOCKS ')
%CYCLE K = 1,1,200; %IF OCC(K)=J %THENSTART; OCC(K)=0; WRITE(K,4)
%FINISH; %REPEAT; ->2
GO: %PRINTTEXT '
INPUT OF CONTROL DATA COMPLETE '
10:COND=NEXTITEM;->11 %IF COND=''''
->F9 %IF COND='?';-"READITEM(COND);->10
11: READLINE(LN,0);SQUASH(LN); %IF LN->('''').ML(1).('''').LN %THEN %C
LN=ML(1) %ELSE ->17
16:
%CYCLE J = 1,1,99; %IF NAME(J) = LN %THENSTART
%IF FS(J,3)#1 %THEN ->17 ;FILENO=J; ->12; %FINISH
%REPEAT; ->17
12: NEWPAGE
PRINTSTRING(LN.'  INPUT STARTED ')
```

```
%BEGIN;
%SHORTINTEGERARRAY  A(1:512*FS(FILENO ,2))
%ROUTINESPEC READDATA(%SHORTINTEGERARRAYNAME  E,%SHORTINTEGER %C
LOGFI,%BYTEINTEGERNAME FAIL,CMODE)
      %INTEGER DEXT
%INTEGER DISK,JJ,KK,NEXT,TIME,V,VA,VB,VC
%STRING (8) %ARRAY  MM(1:15)
JJ=FILENO; FS(JJ,3)=2
READDATA(A,JJ,MORE,FORMAT(JJ))
 110: VC = 1; NEWLINE
%IF  244<=MORE<=255 %THEN ->112
%CYCLE DISK = 1,1,200
%IF OCC(DISK)= JJ %THEN %START
KK = DISK
WRITEDA(12,KK,A((512*-"VC)-511),A(512*VC) )
VC = VC + 1; ->90 %IF VC>FS(FILENO,2); %FINISH
%REPEAT
90: %PRINTTEXT'DATA TRANSFER TO DISC O K'
FS(JJ,3)=3
112:
%END
```

```
%ROUTINE   READDATA(%SHORTINTEGERARRAYNAME  E,%SHORTINTEGER %C
LOGFL,%BYTEINTEGERNAME FAIL,DMODE)
%INTEGER SECT, DSC,NN
%BYTEINTEGER  NO,NP,NQ
%STRING(30) SX,SY
NEXT = 0
FAIL=0; NO = TR
%IF 1<=DMODE<=2 %THENSTART
B:  READLINE(LN,NO)
%IF CHARNO(LN,1) = 39  %THENSTART; FAIL = 254
PRINTSTRING('FILE  HEADING '.LN.'FOUND OUT OF CONTEXT-NO Z TERMINATOR')
->5 ; %FINISH
->EN %IF  LN = 'Z'
->B %IF FAIL = 255
%IF LN->SX.('T').SY %THENSTART;NP=0;->B; %FINISH
%IF NP=1 %AND DMODE=2 %THENSTART;%PRINTTEXT'
EXTRA SCAN IN ETS MODE'; ->B; %FINISH; NP=1
DECOMP(LN,MM,NO)
%IF NO # SCANLENGTH(LOGFL) %THEN %SY"TART
%PRINTTEXT '
WRONG NUMBER OF ITEMS IN LINE ' ; FAIL = 0 ; ->OUT ; %FINISH
%CYCLE SECT = 1,1,SCANLENGTH(LOGFL)
A(NEXT +SECTZ")= INT(S TO R(MM(SECT),SX))
3:  %IF SX= 'F' %THEN %START ; FAIL=2 ; ->OUT ; %FINISH
%REPEAT ; NEXT = NEXT + SCANLENGTH(LOGFL)
%IF    NEXT>512*FS(LOGFL,2) %THENSTART
FAIL= 4;      ->OUT; %FINISH
->B ; %FINISH
%IF DMODE=3 %THENSTART
4: READLINE(LN,NO) ; ->EN %IF LN = 'Z'
%IF LENGTH(LN) # 4*SCANLENGTH(LOGFL) %THEN %START
FAIL=5 ; ->OUT ; %FINISH
%CYCLE SECT = 1,1,SCANLENGTH(LOGFL)
DSC=4*SECT
SY = FROMSTRING(LN,DSC-3,DSC)
A(NEXT + SCANLENGTH(LOGFL))=INT(S TO R(SY,SX));->3 %IF  SX= 'F'
%REPEAT ; NEXT = NEXT +SCANLENGTH(LOGFL); ->4 ; %FINISH
EN: %IF FAIL = 0 %THENSTART; %PRINTTEXT'
DATA INPUT SUCCESSFUL FOR FILENAME ' ; PRINTSTRING(NAME(LOGFL))
%FINISH
```

```
FS(LOGFL,1)=NEXT
NQ=((NEXT-1)//512)+1;FS(LOGFL,2)=NQ
NP=0;       %CYCLE NN=1,1,200; %IF OCC(NN)=LOGFL %THENSTART;NP=NP+1
OCC(NN)=0 %IF NP>NQ;%FINISH; %REPEAT
->5
OUT: TITLE(3,FAIL); %PRINTTEXT'IN DATA INPUT FOR FILENAME'
PRINTSTRING(' '.NAME(LOGFL)); NEWLINE
PRINTSTRING('LINE WAS '. LN)
NO = 0
FAIL = 255 ; ->B
5:  %END
%IF MORE = 254 %THEN ->16 ; ->10
13:
%ROUTINE PACKUP
NEWPAGE
%INTEGER LL,M
SPACES(20)
%PRINTTEXT'CATALOGUE OF CURRENT DATA FILES HELD ON DISC'
PRINTSTRING(' ON '.DATE)
%CYCLE  LL = 1,1,99
NEWLINE; WRITE(LL,2); ->2 %IF FS(LL,3) = 0
SPACES(3); PRINTSTRING(NAME(LL));SPACES(9-LENGTH(NAME(LL)))
->2 %IF NAME(LL) = 'DUMMY'
WRITE(FS(LL,1),4); %PRINTTEXT' ITEMS '
%PRINTTEXT'PRE DATA INPUT' %IF FS(LL,3)=1
%PRINTTEXT'INPUT STAGE    ' %IF FS(LL,3)=2
%PRINTTEXT'REAL VALUES    '%IF FS(LL,3)=4
%PRINTTEXT'COMPLETE       '%IF FS(LL,3) = 3
SPACES(2)
%IF 1<=FS(LL,3)<=2 %THEN %START
%PRINTTEXT'**DELETED**'
NAME(LL)= ''
FS(LL,3) = 0
%CYCLE M=1,1,200; %IF OCC(M)=LL %THEN OCC(M)=0; %REPEAT
%FINISH %ELSE %START
%PRINTTEXT'IN BLOCKS'
BLOCKS=0
%CYCLE M = 1,1,200 ; WRITE (M,3) %IF OCC(M) = LL
BLOCKS=BLOCKS+1 %IF OCC(M) =LL
%REPEAT;SPACES(4*(5-BLOCKS))%IF BLOCKS<5
%PRINTTEXT'LENGTH=';WRITE(SCANLENGTH(LL),2)
%PRINTTEXT' RATE =';WRITE(ISI(LL),2);
%PRINTTEXT' TIME=';M=ISI(LL)*(FS(LL,1)-SCANLENGTH(LL))/SCANLENGTH(LL)
PRINT(INTPT(M/60)+0.6*FRACPT(M/60),2,2)
PRINTSTRING('  '.DT(LL))
%FINISH ; ->3
2:
%IF FS(LL,3) = 0 %THEN %PRINTTEXT '       F R E E '
3:
%REPEAT ; %PRINTTEXT'
ALL INCOMPLETE DATA SETS HAVE BEEN DELETED'
DISC=201;WRITEDA(12,DISC,FS(1,1),FS(100,5))
DISC=202;WRITEDA(12,DISC,BYTEINTEGER(ADDR(NAME(1))), %C
BYTEINTEGER(ADDR(NAME(100))+6))
DISC=203;  WRITEDA(12,DISC,BYTEINTEGER(ADDR(DT(1))),BYTEINTEGER %C
(ADDR(DT(100))+8))
%END
```

```
%BYTEINTEGERMAP  OCC(%INTEGER V)
%INTEGER KA
KA = INTPT((V-1)/2) +1
%RESULT = ADDR(FS(KA,5)) %IF PARITY(V)= -1
 %RESULT = ADDR(FS(KA,5))+1
%END

%ROUTINE DECOMP(%STRING(80) DUP,%STRINGARRAYNAME WD, %C
%BYTEINTEGERNAME DE)
      DE=0; SQUASH(DUP); %IF DUP = '' %THEN -> END
      DE = 1;
1:    WD(DE) = '';
      %IF DUP -> WD(DE).(' ').DUP %THEN ->3 %ELSE -> 4
3: SQUASH(DUP); DF = DE+1; ->1
4:    WD(DE) = DUP
END:
      %END

%ROUTINE SQUASH(%STRINGNAME  S)
1:
%IF FROMSTRING(S,1,1 ) = ' ' %THEN S-> (' ').S %ELSE ->2
->1
2:  %IF FROMSTRING(S,LENGTH(S),LENGTH(S) )=' ' %THEN %START
S = FROMSTRING(S,1,LENGTH(S)-1); ->2; %FINISH
   %END

       %REALFN S TO R(%STRINGNAME S,COND)
       %INTEGER  I,J,K,SIGN
       %REAL Z
       COND='I';      Z= 0; I = 0;SIGN = 1; SQUASH(S)
       ->FAIL %IF S = ''
       %CYCLE J = 1,1,LENGTH(S); K = CHARNO(S,J)
       ->FAIL %UNLESS '0'<=K<='9' %OR  K='.' %OR V"  K='+' %OR K = '-'
       %IF (K='+' %OR K='-') %AND J>1 %THEN ->FAIL
       %IF K = '+' %THEN ->2; %IF K = '-' %THEN %START
       SIGN = -1; ->2;%FINISH
       %IF K = '.' %THEN %START ; COND='R'; ->2; %FINISH
       %IF     COND = 'I' %THEN %START
       Z = 10*Z+(K-'0'); ->2; %FINISH
       %IF COND = 'R' %THEN %START
       I = I + 1; Z = Z +((K-'0')*(10**(-I))); ->2; %FINISH
       ->FAIL
2:
       %REPEAT
FIN:   %RESULT = Z*SIGN
FAIL:    COND='F'; %RESULT = 0
       %END
```

```
      %ROUTINE  READLINE(%STRINGNAME LN , %BYTEINTEGER  TR)
%STRING(72) LO,LP
      %STRING(1)  D ; LN = ''
10:
1:    READITEM(D); ->1 %IF D = H"' '  %OR CHARNO(D,1)=NL
      LN = D
2:    READITEM(D); ->3 %IF CHARNO(D,1) = NL ; LN = LN.D
      ->2
3: %IF LN->LO.('|').LP %THEN ->10
%IF TR # 0 %THEN PRINTSTRING(TOSTRING(NL).LN)
      %END

%INTEGERFN  SECTIME(%REAL U)
%RESULT =  60*INTPT(U) + INT(100*FRACPT(U))      ; %END
17:NEWLINE
PRINTSTRING('WRONG FILENAME '.LN.' WILL NOT BE READ IN')
->10
22:%PRINTTEXT'
**********NO MORE CONTROL DATA WILL BE READ IN ';
23: READLINE(LN,0); DECOMP(LN,ML,NB); %IG"F ML('"1)='GO' %THEN ->GO
->23
F2: TITLE(3,2); ->2
F1:    %PRINTTEXT'****** NO BEGIN COMMAND -PROGRAM ABANDONED'; ->F9
```

3: The ANALYSIS program

```
      %BEGIN
%STRING(12) %ARRAY WORD(1:200)
%STRING(6)%ARRAY  M"NAME(1:140)
%STRING(15)%ARRAY  ML(1:20),CONTRL(1:10)
      %STRING (72) %ARRAY  SLIST(1:50)
%REALARRAY B(1:15),CONSTS(1:80,1:3),RVALS(1:13,1:2)
%SHORTINTEGERARRAY  FS(1:100,1:5)
%INTEGERARRAY  STAD(1:8)
%SHORTINTEGERARRAY  PLIST(1:64,0:7),PAR(1:10)
%SHORTINTEGERARRAY LIB(101:140,1:2),SBLIST(1:2,0:16)
%BYTEINTEGERARRAY KEY(1:15),VARLIST(1:30),INBLK(1:10)
%STRING(80) LN,LINE,MESS,VERS
%REAL RUNTIME
      %INTEGER  J,J1,DISC
%BYTEINTEGER CA,CB,CC,CD,SCANLENGTH,RATE
      %ROUTINESPEC INITIALIZE(%INTEGER Z)
      %ROUTINESPEC DIOLUNITS
%EXTERNALSTRINGFNSPEC  TOSTRING(%INTEGER N)
      %EXTERNALINTEGERFNSPEC CHARNO(%STRINGNAME S, %INTEGER N)
%EXTERNALROUTINESPEC  CLOSEDA(%INTEGER CHANN)
%EXTERNALROUTINESPEC  WRITEDA(%INTEGER CHANN, %INTEGERNAME SECT,%C
%NAME  BEGIN,END)
      %EXTERNALINTEGERFNSPEC LENGTH(%STRINGNAME S)
      %EXTERNALROUTINESPEC OPEN DA(,"%INTEGER  CHANN)
      %EXTERNALROUTINESPEC       READ DA(%INTEGER CHANN,  %C
%INTEGERNAME SECT, %NAME BEGIN,END)
%EXTERNALLONGREALFNSPEC  LOGTEN(%LONGREAL   X)
%EXTERNALREALFNSPEC  RANDOM(%INTEGERNAME I,%INTEGER ("N)
%EXTERNALSTRINGFNSPEC DATE
%EXTERNALLONGREALFNSPEC  CPUTIME
%EXTERNALROUTINESPEC  CLOSESTREAM(%INTEGER STREAM)
%EXTERNALROUTINESPEC  SETMARGINS(%INTEGER STREAM,LH,RH)
%EXTERNALSTRINGFNSPEC  FROMSTRING(%STRINGNAME S,%INTEGER I,J)
      %EXTERNALROUTINESPEC READSTRING(%STRINGNAME S)
      %EXTERNALSTRINGFNSPEC NEXT ITEM
      %EXTERNALROUTINESPEC READ ITEM(%STRINGNAME S)
%INTEGERFNSPEC EXAMINE(%STRING(100) S,%STRING(12) SUBSTR)
      %ROUTINESPEC TITLE(%BYTEINTEGER VAL,A)
%ROUTINESPEC  MCPT(%STRING(100)  SS)
      %ROUTINESPEC PERIODS(%SHORTINTEGERNAME MARKA)
     %ROUTINESPEC CHECKSTATS(%SHORTINTEGERNAME MARKC)
%ROUTINESPEC  SETUP(%STRING(30) S,%SHORTINTEGERNAME  LOGFL)
     %ROUTINESPEC READLINE(%STRINGNAME LN,%BYTEINTEGER TR)
%ROUTINESPEC DISCTRANSFER(%INTEGER K,%ROUTINE R)
%ROUTINESPEC MUDDLE
%INTEGERFNSPEC FR(%INTEGER Z,DIV)
%STRINGFNSPEC  R TO S(%REAL Z,%INTEGER SIGFIGS)
     %ROUTINESPEC SQUASH(%STRINGNAME S)
     %ROUTINESPEC IDENT(%STRINGNAME S,%SHORTINTEGERNAME  VAL)
```

```
%INTEGERFNSPEC  ESCAN(%INTEGER J)
%INTEGERFNSPEC   BSCAN(%INTEGER J)
%INTEGERFNSPEC MEASURE(%INTEGER K)
%INTEGERFNSPEC  GET NO (%STRINGNAME  S)
%ROUTINESPEC  ADDVAR(%INTEGER J)
     %REALFNSPEC  S TO R(%STRINGNAME S,COND)
     %REALFNSPEC TRUETIME(%INTEGER U)
%ROUTINESPEC   FAULT(%INTEGER J,%STRING(80)  AA,AB)
     %INTEGERFNSPEC SECTIME(%REAL U)
%SHORTINTEGERMAPSPEC  FILESTATE
%BYTEINTEGERMAPSPEC STATUS
%BYTEINTEGERMAPSPEC  CCC(%INTEGER V)
%ROUTINESPEC DECOMP(%STRING(80)LN,%STRINGARRAYNAME Z,%BYTEINTEGERNAME %C
NO)
%ROUTINESPEC  TEMPDATA(%BYTEINTEGERNAME  FAIL)
%SWITCH  RA(1:30)
%FAULT 18 ->103
%FAULT 3 ->106
%FAULT 17 ->102
%FAULT 5 ->TIDY
%FAULT 9 ->WINDUP
OPENDA(10);OPENDA(11);OPENDA(12)
STAD(1)=ADDR(WORD(1))
STAD(2)=ADDR(WORD(200))+12; STAD(3)=ADDR(NAME(1))
STAD(4)=ADDR(NAME(140))+7
STAD(5)=ADDR(SLIST(1));STAD(6)=ADDR(SLIST(50))+73
STAD(7) = ADDR(SLIST(28))+72
STAD(8)=ADDR(NAME(101))-1
KEY(6)=1;KEY(3)=1;KEY(9)=0
MCPT('')
READLINE(LN,1);DECOMP(LN,ML,CA)
%IF ML(1) # 'COMMANDS' %THEN ->BOMB
KEY(11)=GETNO(ML(2));%IF (KEY(11)#94 %AND KEY(11)#98 ) %OR %C
10<=KEY(11)<=12 %THEN ->BOMB
RUNTIME=CPUTIME
KEY (7) = KEY(11)
SELECTINPUT(KEY(11))
DISCTRANSFER(1,READDA)
INITIALIZE(22"55); INITIALIZE(0)
VERS='**********DATA LOGGER PROGRAM  MARK 6 VERSION 2 ON 4-75'.  %C
'**********     '.DATE
*
1:NEWPAGE;NEWLINES(2);SPACES(20);PRINTSTRING(VERS);NEWLINE
READLINE(LN,1)
A1: ->WINDUP %IF LN='ENDRUN'
FAULT(104,'NO BEGIN','') %UNLESS      LN='BEGIN'
READLINE(LINE,1);DECOMP(LINE,ML,CA); MCPT(LINE)
%IF ML(1)='DATA' %THENSTART
%IF ML(2) # 'STREAM' %THEN FAULT(110,'','')
KEY(7)=94
RATE=-1
SELECTINPUT(KEY(7)); PAR(1)=100
%IF ML(4)->('ISI*').ML(5) %THEN RATE=GETNO(ML(5))
FAULT(110,ML(5),'') %IF RATE<0
TEMPDATA(CB);KEY(7)=KEY(11)
SELECTINPUT(KEY(11));FAULT(111,LN,'') %IF CB=255
STATUS = 2; ->2; %FINISH
FAULT(105,'','') %IF ML(1)#'FIND'
SETUP(ML(2).' BASIC',PAR(1))
CONTRL(2) = ML(2); STATUS = 2
2: READLINE(LINE,1); MCPT(LINE)
```

```
->RA(PAR(9))
RA(1):J=GETNO(ML(3)); %IF J<6 %THEN FAULT(10,ML(2),ML(3)) %ELSESTART
WORD(J)=ML(2);DISCTRANSFER(1,WRITEDA);%FINISH
RA(2):BIOLUNITS; ->2
RA(3):CHECKSTATS(PAR(8));->2
RA(4):PERIODS(PAR(5));PAR(4)=PAR(5);->2
RA(9): CONTRL(5)=CONTRL(5).'R'; ->2
RA(10):
SETUP(ML(2).' PRD',PAR(10))
%IF PAR(10) # 0 %THENSTART
DISCTRANSFER(PAR(10),READDA);
PAR(5)=LIB(PAR(10),1)
%FINISHELSE FAULT(54,ML(2),'IMPOSSIBLE TO RETRIEVE')
->2
RA(11): CONTRL(5)=CONTRL(5).'L'; ->2
RA(12):->2
RA(13):INITIALIZE(0); ->2
RA(15):CONTRL(5)=CONTRL(5).'P'; ->2
RA(16): PAR(3)=GETNO(ML(2));
%IF PAR(3)<0 %OR 9<PAR(3)<13 %THEN FAULT(33,LINE,'') %ELSE CONTRL(5)= %C
CONTRL(5).'C'; PAR(3)=97; ->2
```

```
RA(19):KEY(3) =0; ->2
RA(20):SETUP(ML(2).' DEL',PAR(10));->2 %IF PAR(10)=0
FAULT(101,ML(2),  '') %IF PAR(1)=PAR(10):NAME(PAR(10))=0"''
%IF 1<=PAR(10)<=100 %THENSTART;FS(PAR(10),3)=0
%CYCLE J = 1,1,200;CCC(J) = 0 %IF CCC(J)=PAR(10)       ;%REPEAT
DISCTRANSFER(2,WRITEDA)
%FINISH
LIB(PAR(10),2)=0 %IF 101<=PAR(10)<=140
DISCTRANSFER(5,WRITEDA);->2
RA(21): LINE->( 'TITLE').MESS; SQUASH(MESS); ->2
RA(22): CB=GETNC(ML(2)); ->2 %IF CB<0
INITIALIZE(CB);STATUS=3 %IF 0<CB<250;->2
RA(23):KEY(3)=1;%IF ML(2)='ALL' %THENSTART
SELECTOUTPUT(9);SETMARGINS(9,1,120);SELECTOUTPUT(99)
KEY(3)=2;       %FINISH; ->2
RA(24):  CA=0; CA = 1 %IF ML(2)='PERIODS'; CA=2 %IF ML(2) = 'STATS'
%IF CA = 0 %THENSTART; FAULT(2,ML(2),''); STATUS = 20; ->17;%FINISH
%CYCLE J = 81+(20*CA),1,101+(20*CA)
%IF LIB(J,2)=0 %THENSTART
LIB(J,2)=1; CB = 4"J;NAME(CB) =ML(3); ->ENTRY; %FINISH; %REPEAT
FAULT(15,'','')
17: READLINE(LN,0); ->17 %UNLESS LN = 'ENDPERIODS' %OR LN = 'ENDSTATS'
->2
ENTRY: PAR(6)=1;
PERIODS(PAR(6)) %IF CA = 1
CHECKSTATS(PAR(6)) %IF CA = 2
%IF (CA=2 %AND PAR(6)>27) %OR (CA=1 %AND PAR(6)>65) %THENSTART
FAULT(17,RTOS(PAR(6),2),''); ->2; %FINISH
SLIST(PAR(6))='ENDFILE' %IF CA =2
LIB(CB,1)=PAR(6);DISCTRANSFER(CB,WRITEDA);NEWLINE
DISCTRANSFER(5,WRITEDA)
CONTRL(5)=CONTRL(5).'L'
PRINTSTRING('PERIODS/STATS LIBRARY FILE '.NAME(CB).' WRITTEN TO FILE ')
WRITE(CB,4); ->2
RA(25): ->2
RA(26): ->2
RA(5): %IF STATUS=20 %THENSTART
%PRINTTEXT'
****NO ANALYSIS  *****'
->TIDY; %FINISH
GO: NEWLINES(2); PRINTSTRING(MESS.'  ALL DATA CORRECT')
->90 %IF PAR(1) = 60
SBLIST(1,0) = RATE
*
KEY(4) = SCANLENGTH
CB=0; CC=0; %CYCLE J = 1,1,SCANLENGTH
%IF VARLIST(J)=1 %THEN ADDVAR(21)
%IF VARLIST(J) = 6 %THENSTART;CB=1; ADDVAR(26); %FINISH
%IF VARLIST(J) = 5 %THENSTART;CC=1;ADDVAR(25);ADDVAR(11); ADDVAR(31)
%FINISH
%REPEAT
%IF CB+CC=2 %THENSTART
ADDVAR(12);ADDVAR(32);%FINISH
%BEGIN
%STRING(72) PSTRING,SMESS
      %STRING(15)  SMALL,TINY
%LONGREALARRAY  S(1:15)
%SHORTINTEGERARRAY  R(1:12),CLIST(1:15),AR(1:3)
%LONGREAL SUMSQ,SUM,STDV,SEM,X,XSQ,TIME,Y
%LONGREAL  MIN,MAX
%SHORTINTEGER JS,JT,JL,JV,SC,N,SJ,VAL,VAR,NOPRDS,NEXT
```

```
%INTEGER I,J,JJ
%REALARRAY A(1:FS(PAR(1),1)),TVL(1:(FS(PAR(1),1)//SCANLENGTH)+5)
%ROUTINESPEC IDENTIFY STATS(%STRINGNAME MM, %SHORTINTEGERNAME  PROC,%C
NOPRDS,VAR,TRANS,SUBSET,DISP,%BYTEINTEGERNAME JOIN,FAIL %C
%SHORTINTEGERARRAYNAME PP)
      %SWITCH ST(1:15)
%ROUTINESPEC FINDPR(%SHORTINTEGER PERNO,%SHORTINTEGERARRAYNAME %C
LIST, %BYTEINTEGERNAME COND)
%ROUTINESPEC ONOFF(%SHORTINTEGER STORE,TYPE,WHICH,%SHORTINTEGERNAME %C
WHEN,C)
%ROUTINESPEC  SUBDEF(%INTEGER TYPE,AFTA,VAL,%SHORTINTEGERNAME %C
RES,COND)
      %INTEGERFNSPEC PERNAMES(%STRINGNAME S)
%BYTEINTEGERFNSPEC INDEX(%SHORTINTEGER U)
%REALFNSPEC ACTUAL(%INTEGER P,VAR,TR)
%REALFNSPEC  GROUP(%SHORTINTEGER  J,VAR,TRANS,NO,SB)
%ROUTINESPEC  LIBRARY LIST
%INTEGERFNSPEC NOW(%SHORTINTEGER Z)
%INTEGERFNSPEC THEN(%SHORTINTEGER  Z)
%REALFNSPEC   CONVERSION(%REALARRAYNAME A,%INTEGER J)
%SHORTINTEGERMAP AS(%INTEGER FL,J)
%OWNSHORTINTEGERARRAY  BUF(1:512)
%OWNINTEGERARRAY  B(1:4)=60,0,0,12
%INTEGER K,L,M,N
K=1+((J-1)//512)
M=J-512*(K-1)
%IF FL=B(1) %AND K=B(3) %THEN ->2
%IF FL=101 %THENSTART
WRITEDA(B(4),B(2),BUF(1),BUF(512));B(1)=102;B(3)=0;->2;%FINISH
%IF B(4)#10 %AND 1<=B(2)<=200 %THEN WRITEDA(B(4),B(2),BUF(1),BUF(512))
%IF FL=100 %THENSTART
B(3)=K; B(2)=K+10; B(4)=10; B(1)=100
READDA(B(4),B(2),BUF(1),BUF(512))
%FINISHELSESTART
B(1)=FL; B(2)=INBLK(K);B(4)=12; B(3)=K
%IF B(2)=-1 %THEN FAULT(115,RTOS(FL,3),RTOS(J,3))
READDA(B(4),B(2),BUF(1),BUF(512))
%FINISH
2: %RESULT=ADDR(BUF(M))
%END
```

```
%ROUTINE  LISTPERIODS
%INTEGER J,K ;%SHORTINTEGERARRAY  ST(1:3);%BYTEINTEGER  COND
KEY(3)=KEY(3)+100
%CYCLE J=1,1,PAR(5)-1
FINDPR(PLIST(J,0),ST,COND);%REPEAT
KEY(3)=KEY(3)-100;NEWPAGE
%PRINTTEXT'
          LIST OF PERIODS AND THEIR POINTERS'
%CYCLE J=1,1,PAR(5)-1;NEWLINE
%CYCLE K=0,1,7;WRITE(PLIST(J,K),8);%REPEAT;%REPEAT
%END
%ROUTINE PRINTCAPTION
%OWNSTRING(7) PY ='PERIOD '
%IF JOIN = 1 %THEN PRINTSTRING(PY.PSTRING)
%IF JOIN = 2 %THEN PRINTSTRING(PY.ML(AY))
TITLE(VAR,1);TITLE(FR(R(4),100),2);NEWLINES(2);%END
%INTEGERFN  POSN(%INTEGER K,VAR)
%INTEGER J
VAR=FR(VAR  ,20) %IF VAR<40
VAR=1 %IF VAR=13;VAR=6 %IF VAR=12;VAR=5 %IF VAR=11
%CYCLE J=1,1,SCANLENGTH
%IF VAR=VARLIST(J) %THENRESULT= BSCAN(K)+J-1
%REPEAT; %RESULT   =0; %END
%INTEGERFN  SET  (%INTEGER  K,SETNO)
%IF SETNO = 0 %THENRESULT = 1
%IF FR(THEN(K)-SBLIST(2,SETNO),SBLIST(1,SETNO))= 0 %THENRESULT = 1 %C
%ELSERESULT = 0
%END
%REALFN  RANDVAL(%LONGREAL M,ST)
%OWNINTEGER I = 231
%LONGREAL  J; %INTEGER L
J = 0; %CYCLE L = 1,1,24;J=J+RANDOM(I,1); %REPEAT
%RESULT=M + ST*((J-12)/SQRT(2)); %END
%BYTEINTEGERFN   INDEX(%SHORTINTEGER U)
%RESULT=VARLIST(FR(U-1,SCANLENGTH)+1)
%END
%REALFN TRANSFORM(%REAL X,%INTEGER TRANS)
%SWITCH  TR(1:10)
%INTEGER K,L
K=TRANS//100;L=TRANS-(100*K)
 >TR(L)
TR(1):%RESULT=X
TR(2):%RESULT=SQRT(X)
TR(3):%RESULT=LOGTEN(X)
TR(5): %RESULT=1/X
TR(4):TR(6):
%IF CONSTS(K,3)=-1 %THENRESULT = -1
X=TRANSFORM(X,R(9))
%IF L = 4 %THENRESULT=50+10*((X-CONSTS(K,1))/CONSTS(K,2))
%IF L = 6 %THENRESULT=(X-CONSTS(K,1))/CONSTS(K,2)
%END
```

```
%LONGREALFN  STANDEV(%LONGREAL SU,SS,N)
%RESULT=SQRT((SS-((SU**2)/N))/(N-1)); %END
%REALFN  CONVERSION(%REALARRAYNAME A,%INTEGER P)
%REAL  X,MA,MB
%INTEGER J
%SHORTINTEGER  M
%OWNSHORTINTEGERARRAY  TABLE(0:15)=0,1007,2009,3008,4010,5013,6003,
6998,8006,8999,1099,2191,3282,4375,5463,6546
%SWITCH CON(1:10)
%IF INDEX(P)=49 %THENRESULT=-1
%IF INDEX(P)=47 %THENSTART;%CYCLE J=1,1,15
%IF TABLE(J)-20<A(P)<TABLE(J)+20 %THENRESULT=J; %REPEAT
%RESULT=0;%FINISH
->CON(INDEX(P))
CON(1): X=A(P)/10;%IF INT(X)=0 %THENRESULT=-1 %ELSERESULT= %C
(10**3)/X
CON(2):  X = (A(P)-B(1))*B(3)/(B(2)-B(1))
%IF B(4)<=X<=B(5) %THENRESULT= X %ELSERESULT=-1
CON(5):  X = (A(P)-B(6))*B(8)/(B(7)-B(6))
%IF B(9)<=X<=B(10)%THENRESULT= X %ELSERESULT=-1
CON(6): %IF P<=SCANLENGTH %THENRESULT=-1
MB=A(P-SCANLENGTH);MA=A(P)
%IF MA>B(12) %THEN MA=B(12)
%IF MB>B(12) %THEN MB=B(12)
X = MA - MB ; %IF X<=-10 %THEN X =(B(12)-B(11))+X
X=60*B(13)*X/(RATE*(B(12)-B(11)))
X=(X*273*B(14))/((B(15)+273)*760)
%IF X<0.5 %OR X>50 %THENRESULT=-1 %ELSERESULT=X
%END
%REALFN ACTUAL(%INTEGER P,VAR,TR)
%REAL  Z; Z=A(P)
%IF 1<=VAR<=6 %THENSTART
1: %IF Z<=0 %THENRESULT=-1 %ELSERESULT=TRANSFORM(Z,TR);%FINISH
%IF  VAR=11 %THENSTART; %IF Z<=0 %THENRESULT=-1 %ELSERESULT= %C
TRANSFORM(60/Z,TR); %FINISH
%IF VAR=12 %THENSTART;Z= TVL(((P-1)//SCANLENGTH)+1)
->1;%FINISH
%IF 47<=VAR<=48 %THENRESULT=Z
%IF 21<=VAR."<=33 %THENRESULT=TRANSFORM(RANC"DVAL(RVALS(VAR-20,1),%C
RVALS(VAR-20,2)),TR)
%RESULT=-1; %END
%INTEGERFN PERNAMES(%STRINGNAME S)
%STRING(4) BA,BB;%INTEGER X;
%IF FROMSTRING(S,1,2) = 'T*' %THENSTART
BB =FROMSTRING(S,3,LENGTH(S));X = 10000+INT(S TO R(BB,BA));
 >2; %FINISH
%IF CHARNO(S,2)='A' %THENSTART;X=CHARNO(S,1)-'A'+1
%UNLESS(   1<=X<=26) %THENSTART; FAULT(8,S,''); %RESULT=-1; %FINISH
BB=FROMSTRING(S,3,LENGTH(S))
%RESULT=11000+(100*X)+INT(S TOR(BB,BA))
%FINISH
X = INT(S TO R(S,BA))
%IF BA='F' %THENRESULT=-1
2: %RESULT = X
%END
```

```
%ROUTINE      SUBDEF(%INTEGER TYPE,AFTA,VAL,%SHORTINTEGERNAME %C
RES,COND)
%SHORTINTEGERARRAY  MINI(1:3);%BYTEINTEGER C
%IF TYPE = 1 %THEN %START
ONOFF(AFTA,FR(VAL,100),INTPT(VAL/100),RES,COND); %FINISH
%IF TYPE=2 %THENSTART
RES=NOW(VAL); RES=ESCAN(RES) %IF COND=4; %FINISH
%IF TYPE=4 %THEN RES= ESCAN(NOW(VAL+(THEN(AFTA))))
%IF TYPE = 5 %THEN RES = VAL
%IF 6<=TYPE<=7 %THEN %START
FINDPR(VAL,MINI,C); COND = -1 %IF C = 0
%IF TYPE=6 %THEN RES=MINI(2) %ELSE RES=ESCAN(MINI(3))+1
%FINISH ; %END
      %ROUTINE FINDPR(%SHORTINTEGER PERNO,%SHORTINTEGERARRAYNAME LIST,%C
      %BYTEINTEGERNAME COND)
%SHORTINTEGER  FA,FB,JJ ; %INTEGER J
      %ROUTINESPEC DEFINITIONS(%SHORTINTEGER  PNAME,%SHORTINTEGERNAME %C
      PBEG,PEND)
COND=1
%IF 10000<=PERNO<=10999 %THENSTART
DEFINITIONS(PERNO,LIST(2),LIST(3)); ->34 %IF LIST(2)=-1
%RETURN;%FINISH
%IF PERNO = 0 %THEN %START
LIST(2)=1;LIST(3)=NEXT;LIST(1)=0
      %RETURN; %FINISH
%IF PERNO>=11000 %AND FR(PERNO,100)#0 %THENSTART
JJ=100*INTPT(PERNO/100);FINDPR(JJ,LIST,COND);->34 %IF COND=0
LIST(2)=LIST(2)+(FR(PERNO,100)-1)*LIST(3)
LIST(3)=LIST(2)+LIST(3)-1; LIST(1)=PERNO ; COND=1;
->34 %UNLESS(1<=LIST(2)<=NEXT) %AND (1<=LIST(3)<=NEXT) %C
%AND LIST(2)<LIST(3); %RETURN; %FINISH
%CYCLE J = 1,1,PAR(5)-1; %IF PLIST(J,0)=PERNO %THENSTART
%IF PLIST(J,6)#0 %THEN ->33
DEFINITIONS(J,PLIST(J,6),PLIST(J,7))
->33; %FINISH ; %REPEAT
PERMESS:FAULT(5,RTOS(PERNO,4),'');COND=0; %RETURN
33: %IF PLIST(J,6)<0 %THEN ->34;LIST(2)=PLIST(J,6);LIST(3)=PLIST(J,7)
LIST(1)=PERNO;%RETURN
34:FAULT(7,RTOS(PERNO,5),'')
COND=0    ;%RETURN
      %ROUTINE DEFINITIONS(%SHORTINTEGER PNAME, %SHORTINTEGERNAME %C
      PBEG,PEND)
      %SHORTINTEGERARRAY D(0:5)
%SHORTINTEGERARRAY  DG(2:5)
%INTEGER  J,DD
%SHORTINTEGER      DFA,AFTA,EA
  EA = 1
%IF 10000<=PNAME<=10999 %THENSTART
J=60*(PNAME-10001);
PBEG=ESCAN( NOW(J))+ 1;PEND =ESCAN(NOW(J+60)); ->CHECK; %FO"INISH
J=PNAME
2:    %CYCLE DD=0,1,5; D(DD)=PLIST(J,DD); %REPEAT
DFA = D(1)
%CYCLE J = 2,1,5;DG(J) =FR(DFA,10);DFA=INTPT(DFA/10);%REPEAT
AFTA = 1
%IF DG(2) = 4 %THEN ->3
```

```
SUBDEF(DG(3),AFTA,D(3),AFTA,EA); ->IMPDEF %IF EA = -1
SUBDEF(DG(2),AFTA,D(2),PBEG,EA)      ; ->IMPDEF %IF EA = -1
->4 %IF PLIST(PNAME,0) >=11000
%IF DG(2) = 1 %AND DG(4) = 0 %THEN %START
EA = 4
ONOFF(PBEG,FR(D(2),100),2,PEND,EA)
->IMPDEF %IF EA = -1 ; ->CHECK; %FINISH
AFTA=PBEG
3:
SUBDEF(DG(5),AFTA,D(5),AFTA,EA); ->IMPDEF %IF EA = -1
EA = 4
SUBDEF(DG(4),AFTA,D(4),PEND,EA); ->IMPDEF %IF EA = -1
%IF DG(2) = 4 %THEN PBEG=BSCAN(PEND-NOW(D(2)))
CHECK: %IF PBEG>=PEND %THEN ->IMPDEF
%IF DG(4) = 7 %THEN  PEND = PEND -1
%UNLESS(1<=PBEG<=NEXT) %AND (1<=PEND<=NEXT) %THEN ->IMPDEF
%RETURN
4:PEND= SCANLENGTH*(D(4)//RATE); %RETURN
IMPDEF:FAULT(25,RTOS(PBEG,4),RTOS(PEND,4));PBEG = -1 ;PEND= -1
%END
35: %END
%ROUTINE  TOTALS(%SHORTINTEGER BEG,END,VAR,SUBST,TR,MODE,GR,%LONG %C
%REALARRAYNAME  C)
%INTEGER G,DIR,GA
%LONGREAL  TIME,GG,HH,ZERO,LAST,M,STD
%IF BEG>END %THEN DIR=-SCANLENGTH %ELSE DIR=SCANLENGTH
GA=SCANLENGTH*GR*(SBLIST(1,SUBST)/RATE)
ZERO=THEN(BEG)
%CYCLE G=POSN(BEG,VAR),DIR,POSN(END,VAR)
%IF SET(G,SUBSY"T)= 1 %THENSTART
GG=GROUP(G,VAR,TR,GR,SUBST)
->F %IF GG<0
C(1)=C(1)+GG;CO"(2)=C(2)+(GG**2); C(3)=C(3)+1
MIN=GG %IF GG<MIN;MAX=GG %IF GG>MAX
%IF MODE=1 %THENSTART;TIME=(THEN(G)-ZERO)/60
C(4)=C(4)+(GG*TIME);C(5)=C(5)+TIME;C(6)=C(6)+(TIME**2)
%FINISH
->3
F:%UNLESS (G<=SCANLENGTH %AND INDEX(G)=6) %THEN C(7)=C(7)+1
MODE=0 %IF MODE=3
3: %IF MODE >1 %THENSTART
%IF GG<0 %THEN HH=TRANSFORM(RANDVAL(C(13),C(14)) ,TR) %ELSE HH=GG
C(8)=C(8)+HH;C(9)=C(9)+(HH**2);C(10)=C(10)+1
%IF C(10) # 1 %THEN C(11)=C(11)+((HH-LAST)**2); LAST=HH; %FINISH
%FINISH
%IF 0<GR<1000 %THEN G=G+GA
%UNLESS G<=END %AND G>=BEG %AND 1<=G<=NEXT %THENRETURN
2: %REPEAT; %END
```

```
      %ROUTINE ONOFF(%SHORTINTEGER STORE,TYPE,WHICH,%SHORTINTEGERNAME %C
      WHEN,C)
%STRING(4)  G
%SHORTINTEGER KK,LL,VC
%INTEGER J,I;I=C;C=0
%IF STORE>NEXT %THEN ->19
%IF WHICH = 2 %THEN G = 'OFF' %ELSE G = 'ON'
KK=FR(TYPE,50);%IF TYPE<50 %THEN VC=47   %ELSE VC=48
      %IF WHICH = 2 %THEN %START
ONOFF(STORE,TYPE,1,LL,C);STORE=LL
%IF C=-1 %THEN ->19;%FINISH
%CYCLE J=STORE,1,NEXT; %IF INDEX(J) = VC %THENSTART
C=INT(A(J));->2 %IF (C=KK %AND WHICH=1) %OR (C#KK %AND WHICH=2)
%FINISH;%REPEAT
2:->19 %IF ESCAN(J)=NEXT %AND WHICH=1
%IF  I=4 %THEN WHEN = BSCAN(J)-1 %ELSE WHEN=BSCAN(J)
%RETURN
19: FAULT(6,RTOS(KK,4),G); C = -1
   %END
%INTEGERFN PHYSVAR
%INTEGER J
1: VAR=VAR+1; %IF VAR>=7 %THENRESULT=50
%CYCLE J=1,1,SCANLENGTH;%IF VARLIST(J)=VAR %THENRESULT=VAR;%REPEAT
->1
%END
%ROUTINE UPDATE
AY = AY + 1; R(2) = CLIST(AY); FINDPR(R(2),AR,SC); %END
%REALFN GROUP(%SHORTINTEGER J,VAR,TRANS,NO,SB)
%LONGREALARRAY SP(1:7)
%IF NO =0 %THENRESULT=ACTUAL(J,VAR,TRANS)
NO=FR(NO,10000")
SP(1)=0;SP(2)=0;SP(3)=0;SP(7)=0
TOTALS(J,J+(NO*SCANLENGTH),VAR,SB,TRANS,08",C,SP)
%IF SP(3)= 0 %THENRESULT=-1 %ELSERESULT=SR"P(1)/SP(3); %END
%ROUTINE AUTOCOVAR(%SHORTINTEGER FROM,TO,INT,ARNO,VAR,TR,GRP)
%LONGREAL SUM,AVE,COV      ,SD
%REALARRAY BB(1:ARNO)
%INTEGER J,K,L,LA
K=1; AVE=0    ;SD=0
%CYCLE J=FROM,INT,TO;X=GROUP(J,VAR,TR,GRP,0)
%IF X=-1 %THEN X=ACTUAL(J,VAR+20,TR)
AVE=AVE+X;SD=SD+(X**2);BB(K)=X;K=K+1;%REPEAT
K=K-1;SD=STANDEV(AVE,SD,K);  AVE=AVE/K
LA=K-1
NEWLINES(3); %PRINTTEXT' AUTOCOVARIANCES FOR '; PRINTCAPTION; NEWLINE
%PRINTTEXT'  MEAN = '; PRINTFL(AVE,4); %PRINTTEXT' N ='; WRITE(K,4)
%PRINTTEXT'
 LAG    COVARIANCE    AUTOCORRELATION'
%CYCLE J=1,1,LA;SUM=0;
%CYCLE L=1,1,K-J+1;SUM=SUM+(BB(L)-AVE)*(BB(L+J-1)-AVE);%REPEAT
COV=SUM/(K-J+1);NEWLINE
WRITE(J- 1,3);SPACES(4);PRINTFL(COV,4);SPACES(2);PRINTFL(COV/(SD**2),4)
%REPEAT
%END
```

```
MCPT('R.I.D. BEGINS')
SMESS='' ;NEXT=FS(PAR(1),1)
%CYCLE J=1,1,NEXT
A(J)=AS(PAR(1),J);
%REPEAT
%IF EXAMINE(CONTRL(5),'R')=1%THEN CA=1 %ELSE CA=0
%IF FILESTATE #4 %THENSTART
%CYCLE J=NEXT,-1,1;A(J)=CONVERSION(A,J)
%IF CA=1 %THENSTART;
%IF INDEX(J)#1 %THEN AS(PAR(1),J)=INT(A(J)*100) %ELSE AS(PAR(1),J)=%C
INT(A(J)*1000)-30000
%FINISH
%REPEAT
%IF CA=1 %THENSTART;FILESTATE=4;DISCTRANSFER(2,WRITECA);%PRINTTEXT'
REAL VALUES FILED ON DISC';%FINISH
%FINISHELSESTART
%CYCLE J=NEXT,-1,1
%IF INDEX(J) #1  %THEN A(J) =A(J)/100  %ELSE A(J)=(A(J)+30000)/1000
%REPEAT
%FINISH
I=AS(101,1)
MCPT('R.I.D. ENDS')
%IF POSN(1,12)#0 %THENSTART;TVL(1)=-1;TVL(2)=-1
%CYCLE J=1+2*SCANLENGTH,SCANLENGTH,BSCAN(NEXT)
I=POSN(J,6);JJ=POSN(J,5);JV=1+(J//SCANLENGTH)
Y=GROUP(I,6,1,-2,0);X=GROUP(JJ,5,1,-2,0)
%IF Y=-1 %OR X=-1 %THEN TVL(JV)=-1%ELSE TVL(JV)=1000*Y/X;%REPEAT;%FINISH
MCPT ('TV CALC')
      S1 = 1
%CYCLE JJ=1,1,13
%IF POSN(1,JJ)#0 %THENSTART
%CYCLE 4"J=1,1,15;S(J)=0; %REPEAT
MAX=-10**50;MIN=10**50
TOTALS(1,NEXT,JJ,0,1,0,0,S)
%IF S(3)>2 %THENSTART
RVALS(JJ,1)=S(1)/S(3);CONSTS(JJ,1)=S(1)/S(3)
RVALS(JJ,2)-STANDEV(S(1),S(2),S(3))
CONSTS(JJ,2)=RVALS(JJ,2);CONSTS(JJ,3)=JJ+100
%FINISH;%FINISH;%REPEAT
MCPT('CALC CONSTS')
%IF EXAMINE(CONTRL(5),'C')=1 %THENSTART;SELECTOUTPUT(PAR(3))
PRINTSTRING(NAME(PAR(1)));SPACES(7-LENGTH(NAME(PAR(1))))
%CYCLE J=1,1,SCANLENGTH
%IF 47<=VARLIST(J)<=50 %THEN JJ=110+VARLIST(J) %ELSE JJ=120+VARLIST(J)
PRINTSTRING(WORD(JJ));SPACES(8);%REPEAT;NEWLINE
%CYCLE JJ=1,SCANLENGTH,BSCAN(NEXT)
PRINT(TRUETIME(THEN(JJ)),2,2);SPACE
%CYCLE I=1,1,SCANLENGTH
PRINT(A(I+JJ-1),3,4);SPACE;%REPEAT;NEWLINE
%REPEAT;SELECTOUTPUT(99)
MCPT('FINISH CARDS')
%PRINTTEXT'
DATA PUNCHED ONTO CARDS SUCCESSFULLY';%FINISH
%IF EXAMINE(CONTRL(5),'I')=1 %THEN LIBRARYLIST
%IF EXAMINE(CONTRL(5),'P') =1 %THEN LISTPERIODS
MCPT('BEGIN STATS')
1:
```

```
%CYCLE J = 1,1,12; R(J) = 0; %REPEAT
LN=SLIST(S1)
VAR = 0
2:
%IF LN ='ENDFILE' %THENSTART
DISCTRANSFER(3,READDA); S1 =PAR(10);LN=SLIST(S1); %FINISH
%IF EXAMINE(LN,'CONTINUE')=1 %THENSTART
DECOMP(LN,ML,AZ); SETUP(ML(3).' ST',JS)
->BACK %IF JS=0;
PAR(10)= S1+1;
DISCTRANSFER(3,WRITEDA)
DISCTRANSFER(JS,READDA); S1=1
->1
%FINISH
%IF LN='ENDSTATS' %THEN ->RET
NOPRDS = 1
IDENTIFY STATS(LN,R(1),NOPRDS,R(3),R(4),R(5),JV,JOIN,FAIL,CLIST)
MCPT(LN.' IDENT')
%IF S1 = 1 %OR SMESS # '' %THENSTART
NEWPAGE; PRINTSTRING(MESS.'  '.SMESS)''; SMESS=''; %FINISH
%IF FAIL = 1 %OR FAIL = 2 %THEN ->BACK
NEWLINES(3);PRINTSTRING(LN)
%IF R(3)=50 %THEN C"VAR=PHYSVAR %ELSE VAR=R(3)
%IF POSN(1,VAR)=0 %THENSTART
FAULT(24,WORD(VAR+160),'NOT MEASURED');->PH;%FINISH
AY = 0
6:  UPDATE ; ->14 %IF SC = 0
GO:
%CYCLE J=1,1,15; S(J)=0; %REPEAT
%IF R(3)=50 %AND VAR=1 %AND ALLRAW=1 %THEN R(4)=3
      ->ST(R(1))
ST(1)    :
%CYCLE  J = AR(2),1,AR(3)
%IF INDEX(J)=VAR %THEN A(J)=-1
%REPEAT ;
NEWLINE;PRINTSTRING(WORD(VAR+160). ' DELETED IN PERIOD ')
WRITE(R(2),4); ->5
ST(4): NEWLINES(5)
PRINTSTRING('REGRESSION OF '.WORD(VAR+160).%C
' ON TIME IN MINUTES FOR PERIOD '.ML(AY))
TOTALS(AR(2),AR(3),VAR,R(5),R(4),1,R(6),S)
->44 %IF S(3)<3
S(6)=S(6)-(S(5)**2/S(3))
S(2) =S(2) - (S(1)**2/S(3))
S(4)=S(4)-(S(5)*S(1)/S(3))
NEWLINES(2)
NEWLINE; %PRINTTEXT'          REGRESSION COEFFICIENT = ':
PRINTFL(S(4)/S(6),6)
%PRINTTEXT'           CONSTANT ='
PRINTFL((S(1)/S(3))-((S(4)/S(6))*(S(5)/S(3))),6)
NEWLINE
```

```
%PRINTTEXT '
NUMBER OF USABLE READINGS    =     '; PRINT(     S(3),3,0)
%PRINTTEXT'
NUMBER OF UNUSABLE READINGS =     '; PRINT(S(7),3,0)
NEWLINES(2)
%PRINTTEXT'
          ANALYSIS OF  VARIANCE
          SOURCE OF VARIANCE              SS                    MS       DF  '
NEWLINE; %PRINTTEXT'
          REGRESSION          '
S(7) - S(4)**2/S(6)
PRINTFL(S(7),6); SPACES(4);PRINTFL(S(7),6); SPACES(7); WRITE(1,1)
NEWLINE; %PRINTTEXT'
          RESIDUAL            '
PRINTFL(S(2)-S(7),6); SPACES(4)
PRINTFL((S(2)-S(7))/S"(S(3)-2),6); SPACES(6); WRITE(INT(S(3)) -2,2)
NEWLINE; %PRINTTEXT'
          TOTAL               '
PRINTFL(S(2),6); SPACES(23); PRINT(S(3)-1,2,0)
NEWLINES(2); %PRINTTEXT'          F  VALUE =  '
PRINT(S(7)/((S(2)-S(7))/( S(3)-2)),3,3)
->5
44: %PRINTTEXT'
          NOT ENOUGH READINGS FOR REGRESSION ANALYSIS '; ->5
ST(2): NEWLINES(5)
21:SPACES(30);%PRINTTEXT' DATA TABLE FOR ';PRINTCAPTION
22:  %CYCLE J = 1,1,6; PRINTSTRING('  TIME  VARIABLE    '); %REPEAT
NEWLINES(2);SD=0
%CYCLE J=POSN(AR(2),VAR),SCANLENGTH,POSN(AR(3),VAR); ->24 %IF SET %C
(J,R(5)) = 0
X-GROUP(J,VAR,R(4),R(6),R(5))
SD=SD+1
PRINT(TRUETIME(THEN(J)),2,2); SPACE
%IF INT(X) # -1 %THEN PRINTFL(X,4) %ELSEPRINTTEXT' **********'
SPACES(2)
%IF FR(SD,6)=0 %THEN NEWLINE
24:
%IF 0<R(6)<1000 %THEN J = J +(SCANLENGTH*R(6)*SBLIST(1,R(5))/RATE)
->5 %IF J>POSN(AR(3),VAR)
%REPEAT          : ->5
ST(3):NEWLINES(4);SPACES(30)
%PRINTTEXT'TABLE OF GENERAL STATISTICS FOR '; PRINTCAPTION
MIN=10**50; MAX=-10**50
32:
TOTALS(AR(2),AR(3),VAR,R(5),R(4),0,R(6),S)
%IF (JOIN = 1 %AND AY<NOPRDS) %THENSTART
UPDATE; ->14 %IF SC = 0; ->32; %FINISH
SUM=S(1);SUMSQ=S(2);N=INT(S(3));->36 %IF N<3
XSQ =  SUMSQ - (SUM**2/N)
STDV=STANDEV(S(1,"),S(2),S(3))
%PRINTTEXT'            TOTAL       MEAN          ST.DEV          S.E.M'
%PRINTTEXT'          XSQ            DEV X SQ       N'
```

```
35: NEWLINES(2); SPACES(8)
      PRINTFL(SUM,4); SPACE
      PRINTFL(SUM/N%",4); SPACES(2)
PRINTFL(STDV,6); SPACES(2)
PRINTFL(STDV/SQRT(N),4); SPACE
      PRINTFL(SUMSQ,6); SPACE
      PRINTFL(XSQ,6); SPACE
WRITE(N,3)
->81 %IF R(1)=8
%PRINTTEXT'
        MINIMUM= ';PRINTFL(MIN,4);%PRINTTEXT' MAXIMUM = '
PRINTFL(MAX,4);NEWLINE;SPACES(7);
PRINT(S(7),3,0);%PRINTTEXT' UNACCEPTABLE READINGS'     ;->5
36:      %PRINTTEXT'**********NO ACCEPTABLE READINGS IN THIS PERIOD'
->5
ST(5):N=INTPT((THEN(AR(3))-THEN(AR(2))+RATE)/(5*RATE))
JU=1;SBLIST(2,16)=THEN(AR(2))
NEWLINES(5)
PRINTSTRING('
TIME SERIES ANALYSES FOR '); PRINTCAPTION; %PRINTTEXT'
FREQUENCY DL.SQ.X      VARIANCE    V.N.R   N'
51:SBLIST(1,16)=JU*RATE
%CYCLE J =1,1,15; S(J) = 0; %REPEAT
TOTALS(AR(2),AR(3),VAR,16,R(4),3,0,S)
%IF S(3)<=2 %THENSTART; %PRINTTEXT'
***NOT ENOUGH READINGS FOR THIS ANALYSIS ***'; ->5; %FINISH
%IF S(7)>0 %THENSTART
S(13)=S(1)/S(3);S(14)=STANDEV(S(1),S(2),S(3))
%CYCLE J=1,1,11;S(J)=0;%REPEAT
TOTALS(AR(2),AR(3),VAR,16,R(4),2,0,S)
%FINISH
S(8)=STANDEV(S(8),S(9),S(10))**2
S(11)=S(11)/(S(10)-1); NEWLINE; SPACES(2)
WRITE(SBLIST(1,16),3)
SPACES(2); PRINTFL(S(11),3); SPACES(2); PRINTFL(S(8),3)
SPACES(2); PRINT(S(11)/S(8),2,3); SPACES(2); WRITE(INT(S(10)),3)
%IF S(7)#0 %THEN PRINTSTRING('  '.RTOS(S(7),2).'  SUBSTITUTE VALUES')
JU=JU+1
->5 %IF JU > N; ->51
ST(6): JT=POSN(AR(2),VAR);JU=POSN(AR(3),VAR);JV=SCANLENGTH
%IF R(5)# 0 %THENSTART
61: %IF SET(JT,R(5))=0 %THEN JT=JT+SCANLENGTH
%IF SET(JU,R(5))=0 %THEN JU=JU-SCANLENGTH
->61 %UNLESS SET(JT,R(5))=1 %AND SET(JU,R(5))=1
%FINISH
JV=(SBLIST(1,R(5))//RATE)*SCANLENGTH
%IF JT>=JU %THENSTART;FAULT(29,'','');->BACK;%FINISH
JS=((JU-JT)//JV)+10
AUTOCOVAR(JT,JU,JV,JS,VAR,R(4),R(6)); ->5
```

```
ST(8): %IF R(3)=50 %THENSTART;FAULT(26,LN,'');->5;%FINISH
NEWLINES(2);JOIN=1;->32
81:CONSTS(R(7),1)=SUM/N;CONSTS(R(7),2)=STDV
CONSTS(R(7),3)=VAR+100*R(4); ->BACK
ST(10): NEWLINES(5); PRINTSTRING('
LIST OF MINIMA AND MAXIMA FOR '); PRINTCAPTION
JT =1
102: %CYCLE J=POSN(AR(2),VAR),SCANLENGTH,POSN(AR(3),VAR); SD=0
%IF SET(J,R(5))= 1 %THENSTART
X=ACTUAL(J,VAR,R(4));->100 %IF X=-1
%IF JT<3 %THENSTART;S(JT)=X;S(JT+3)=TRUETIME(THEN(J))
JT=JT+1; %FINISHELSESTART
S(3)=X; S(6)=TRUETIME(THEN(J)); JT=JT+1
%IF S(1)<S(2) %AND S(2)>S(3) %THENSTART ;PRINTSTRING('
MAX AT  ');PRINT(S(5),2,2);     PRINTFL(S(2),3)
SD=1; %FINISH
%IF S(1)>S(2) %AND S(2)<S(3) %THENSTART; PRINTSTRING('
MIN AT  ');PRINT(S(5),2,2) ; PRINTFL(S(2),3)
SD=1; %FINISH
%IF VAR=1 %AND R(4) = 3 %ANS"D S(2)-S(1)>0.003  %THENSTART
%IF SD=0 %THENSTART;NEWLINE;SPACES(8);PRINT(S(5),2,2);PRINTFL(S(2),3)
%FINISH
PRINTSTRING(' S.F ?'); %FINISH
S(1)=S(2) ; S(2)=S(3); S(4)=S(5); S(5)=S(6); %FINISH
%FINISH
100: %REPEAT
%IF JOIN=1 %AND NOPRDS>AY %THENSTART
UPDATE ; ->14 %IF SC = 0; ->102; %FINISH; ->5
5:MCPT(LN.'  '.ML(AY).'  '.WORD(VAR+120))
%IF AY<NOPRDS %THENSTART
UPDATE ; ->14 %IF SC = 0; ->GO; %FINISH
PH:AY=0; %IF R(3)#50  %THEN ->BACK
%IF R(3)=50 %AND VAR=1 %AND ALLRAW=1 %THEN R(4)=1
VAR = PHYSVAR ; ->6 %IF VAR<=6
BACK:S1=S1+1;%IF S1>50 %THEN MUDDLE;->1
14: NEWLINE; PRINTSTRING('COMMAND '.LN. %C
' NOT EXECUTED - ILLEGAL PERIOD NO ');WRITE(R(2),6); ->5
RET: NEWLINES(5); %PRINTTEXT'
**********ALL  STATISTICAL TESTS COMPLETED FOR THIS SET OF DATA********'
```

```
%ROUTINE IDENTIFY STATS(%STRINGNAME MM,%SHORTINTEGERNAME PROC, %C
NOPRDS,VAR,TRANS,SUBSET,DISP,%BYTEINTEGERNAME JOIN,FAIL %C
%SHORTINTEGERARRAYNAME PR)
%STRING(150)  T1,T2,LN
%STRING(1) COND;%INTEGER J;%BYTEINTEGER AZ
LN = MM ; JOIN = 0 ; SUBSET = 0 ; FAIL = 0
T1='';T2='';DISP=0
%IF EXAMINE(LN,'ALL') *EXAMINE(LN,'RAW')=1 %THEN ALLRAW = 1%C
%ELSE ALLRAW = 0
%IF LN ->SMALL.('*MESSAGE').SMESS %THENSTART; SQUASH(SMESS)
FAIL = 2; %RETURN ; %FINISH
%IF LN->T2.('SUBSET*').T1.(' ').LN %THENSTART;LN=T2.' '.LN;SQUASH(T1)
SUBSET=GETNO( T1);->F2 %IF SUBSET<0;%FINISH
%IF LN->T2.('SMOOTH*').T1.(' ').LN %THENSTART;LN=T2.' '.LN;SQUASH(T1)
R(6)=1000+ INT(STOR(T1,COND));->F2 %IF COND = 'F'
%FINISH
%IF LN->T2.('GROUP*' ).T1.(' ').LN %THENSTART;LN=T2.' '.LN;SQUASH(T1)
R(6)=GETNO(T1);->F2 %IF R(6)<0;
%FINISH
%UNLESS LN->T1.('(').PSTRING.(')').T2 %THEN ->F6
T1=T1.' '.T2
DECOMP(T1,ML,AZ)
%IF ML(1) ->ML(1).('*').T2 %THENSTART
->F1 %UNLESS ML(1) = 'NORMS';  |OR ANY OTHER THAT NEEDS A SUB PARAM
R(7)=GETNO(T2);->F2 %IF R(7)<0;%FINISH
IDENT(ML(1),PROC);->F9 %IF PROC<90 %OR PROC>105
PROC = PROC -89
IDENT(ML(2),VAR);VAR=MEASURE(VAR);->F3 %UNLESS VAR=50 %OR 1<=VAR<=33
%IF ML(3)->ML(3).('*').T2 %THENSTART
->F1 %UNLESS ML(3)='TSCORE' %OR ML(3)='STSCORE'
R(8)=GETNO(T2);->F2 %IF R(8)<0
R(9) = INTPT(CONSTS(R(8),3)/100)
%FINISH
IDENT(ML(3),TRANS); TRANS = TRANS -109; ->F5 %IF 1>TRANS>10
TRANS=TRANS+100*R(8)
T1 = PSTRING
SQUASH(T1); %IF T1->T2.('.').T1  %THEN %START
SQUASH(T2)    ; JOIN = 1 %IF T2='C';JOIN=2 %IF T2 = 'I'
JOIN=3 %IF T2 ='N'
->F1 %IF JOIN=0; %FINISH %ELSE  JOIN =2
SQUASH(T1);DECOMP(T1,ML,AZ);->F8 %IF AZ<1 %OR AZ>15
->NORMS %IF JOIN=3
%CYCLE J = 0"1,1,AZ
PR(J)=PERNAMES(ML(J));->F4 %IF PR(J)=-1
%REPEAT;NOPRDS=AZ;->EN
NORMS:FAIL=2;->F7 %UNLESS PROC=8 %AND 1<=R(7)<=80
CONSTS(R(7),1)=STOR(ML(1),COND); ->F7 %IF COND='F'
CONSTS(R(7),2)=S TO R (ML(2),COND); ->F7 %IF COND = 'F'
CONSTS(R(7),3)=VAR+(100*TRANS)
->EN
F1:F2:F4: FAULT(2,MM,''); ->FL
F3:  FAULT(12,T1,''); ->FL
F5:FAULT(11,T2,''); ->FL
F6: FAULT(19,MM,''); ->FL
F7: FAULT(18,LN,''); ->FL
F8:FAULT(16,RTOS(AZ,3),'');->FL
F9:FAULT(23,ML(1),'')
FL:     FAIL = 1
EN:
%IF R(6)# 0 %THEN R(6)=R(6)-1
      %END
```

```
      %INTEGERFN THEN(%SHORTINTEGER Z)
%RESULT=RATE*((Z-1)//SCANLENGTH)
      %END
      %INTEGERFN NOW(%SHORTINTEGER Z)
%RESULT=1+((Z//RATE)*SCANLENGTH)
      %END
%ROUTINE LIBRARYLIST
%INTEGER J,K
NEWPAGE
PRINTSTRING('  LIST OF LIBRARY FILES  ON  '.DATE)
NEWLINES(2)
%CYCLE  J =101,1,140;%IF LIB(J,2)#0 %THENSTART
WRITE(J,4);PRINTSTRING('    '.NAME(J))
WRITE(LIB(J,1),5);NEWLINE;%FINISH;%REPEAT
%END
      %END
90:NEWLINES(2);
PRINTSTRING('CPU TIME FOR '.CONTRL(2).' WAS ');
PRINT(CPUTIME-RUNTIME,3,6); RUNTIME= CPUTIME; MCPT('END ANALYSIS')
INITIALIZE(255); ->1
102: FAULT(102,'','')
103: FAULT(103,'','')
106: FAULT(106,'','')
TIDY : %PRINTTEXT      '
**********CURRENT  ANALYSIS ABANDONED    **********'
NEWPAGE
STATUS=255;INITIALIZE(255)
200:READLINE(LN,0)
%UNLESS LN='BEGIN' %OR LN='ENDRUN' %THEN ->200
STATUS=1;->A1
WINDUP: %IF STATUS =253 %THEN ->91
%IF KEY(7)#KEY(11) %THENSTART;KEY(7)=KEY(11);SELECTINPUT(KEY(11))
FAULT(113,'','')
%FINISH
MCPT('ENDJOB');
%IF KEY(3)=2 %THENSTART;CLOSESTREAM(9);SELECTINPUT(9)
STATUS=253;NEWPAGE;PRINTSTRING('LOG OF RUN ON  '.DATE);NEWLINES(3)
51:READSYMBOL(J);PRINTSYMBOL(J);-)">51;%FINISH
91:
CLOSEDA(10);CLOSEDA(11);CLOSEDA(12)
NEWLINES(7)
%PRINTTEXT'
**********    E N D  O F  J O B **********
'  ; %STOP
BOMB:PRINTSTRING(LINE.'  ILLEGAL CHANNEL ASSIGNATION COMMAND '.%C
'- JOB ABANDONED  ');%STOP
```

```
%ROUTINE      TEMPDATA(%BYTEINTEGERNAME  FAIL)
%SHORTINTEGERARRAY C(1:2560)
%INTEGER J,K,L
%STRING(1) M
1: M = NEXTITEM; %IF M = '''' %THEN ->2
READITEM(M) ; ->1
2: READSTRING(CONTRL(2)); NAME(100) =CONTRL(2); J=0; FAIL=0
NEWLINE; PRINTSTRING(    ' DATA INPUT FOR FILENAME '.CONTRL(2))
3:  READLINE(LN,0); ->3 %IF EXAMINE(LN,'T')= 1
->98 %IF LN = 'Z'
DECOMP(LN,ML,CA); SCANLENGTH=CA %IF J = 0
->99 %IF LENGTH(ML(1))>7 %OR CA#SCANLENGTH
%CYCLE K=1,1,CA; C(J+K)=GETNO(ML(K));->99 %IF C(J+K)< 0
%REPEAT
J =J + K; ->99 %IF J>2560; ->3
98:  %CYCLE L = 1,1,INTPT((J-1)/512)+ 1
K=10+L;INBLK(L)=K;
WRITEDA(10,K,C(512*(L-1)+1),C(512*L))
%REPEAT
FS(100,1) = J; FS(100,2) = L
FS(100,3)= 3
WRITE(1,3): %PRINTTEXT'  BLOCKS'
%RETURN
99:  FAIL=255
%END
%ROUTINE IDENT(%STRINGNAME S,%SHORTINTEGERNAME VAL)
     %INTEGER J
1: %CYCLE J = 1,1,200; ->2 %IF S=WORD(J); %REPEAT
FAULT(4,S,'');VAL=0;%RETURN
2:    VAL = J
     %END
%ROUTINE DEFN(%SHORTINTEGERNAME TYPE,VAL,%BYTEINTEGERNAME POINT)
%STRING(1) C
POINT=POINT+1
%IF ML(POINT) -'PERIOD' %THEN %START;POINT =POINT+1
VAL=INT(STOR(ML(POINT),C))
POINT=POINT+1
%IF ML(POINT)='START' %THEN TYPE =6; %IF ML(POINT)='FINISH' %THEN TYPE=7
->9 %UNLESS 6<=TYPE<=7 ; ->END ; %FINISH
%IF ML(POINT) = 'DURATION' %THENSTART; POINT=POINT +1;
VAL=SECTIME(STOR(ML(POINT),C))
TYPE=4; ->END; %FINISH
%IF ML(POINT)='ADDR' %THEN %START ; POINT = POINT + 1
VAL=INT(STOR(ML(POINT),C))
TYPE=5; ->END; %FINISH
%IF ML(POINT)= 'TIME' %THEN %START
POINT = POINT + 1
VAL=SECTIME(STOR(ML(POINT),C))
TYPE=2; ->END; %FINISH
%UNLESS ML(POINT)= 'CA' %OR ML(POINT)='CB' %THEN ->9
TYPE = 1
%IF ML(POINT)='CA' %THEN VAL =0 %ELSE VAL = 50 ; POINT = POINT+1
VAL=VAL+INT(STOR(ML(POINT ),C))
POINT = POINT+1;
->9 %IF ML(POINT)#'ON' %AND ML(POINT) #'OFF'
%IF ML(POINT)='ON' %THEN VAL=VAL+100 %ELSE VAL=VAL+200
->END
9:   TYPE=-1
FAULT(2,ML(POINT) ,'');POINT=POINT+1;%RETURN
END: POINT=POINT+1; ->9 %IF C='F'
     %END
```

```
%ROUTINE PERIODS (%SHORTINTEGERNAME MARKA)
%INTEGER J,C,F,E
%BYTEINTEGER PC,PA
%SHORTINTEGERARRAY  D(0:5),ZZ(1:4)
%STRING(1)  COND
1:    READLINE(LN,KEY(3))
%IF EXAMINE(LN,'GO')=1 %OR EXAMINE(LN,'BEGIN')=1%THEN MUDDLE
%CYCLE J = 0,1,5 ; D(J) =C ; %REPEAT
%CYCLE J = 1,1,4 ; ZZ(J) = 0 ; %REPEAT
F = MARKA
%IF LN='ENDPERIODS' %THENRETURN
DECOMP(LN,ML,PA)
%IF ML(1)='MARKER' %THENSTART
C=1+CHARNO(ML(2),1)-'A' ; ->F1  %UNLESS 1<=C<=26
D(0)=11000+(100*C); ->8; %FINISH
%IF  EXAMINE(ML(1),'DEF') # 1 %THEN ->F1
E=GETNO(ML(2));->F1 %IF E<0
%CYCLE J=1,1,MARKA
%IF PLIST(J,0)=E  %THEN F=J; %REPEAT
%IF F>64 %THENSTART; FAULT(17,'',''); ->1; %FINISH
D(0)=E
8:
%IF ML(3) #'FROM' %THEN ->F1;PC=3;DEFN(ZZ(1),D(2),PC);
%IF ML(PC)='AFTER' %THEN  DEFN(ZZ(2),D(3),PC)
->K"51 %IF ML(1) ='MARKER'
%IF ML(PC)='UNTIL' %THEN  DEFN(ZZ(3),D(4),PC)
%IF ML(PC) = 'END' %THEN -> 50 ; |SHORT FORM OF DEFINITION
%IF ML(PC)='AFTER' %THEN  DEFN(ZZ(4),D(5),PC)
50: ->F1 %IF ML(PC) # 'END'
->F1 %IF ZZ(2)=4 %OR ZZ(4) = 4
%IF ZZ(1)<0 %OR ZZ(2)<0 %OR ZZ(3)<0 %OR ZZ(4)<0 %THEN ->F1
%IF (ZZ(1)#1 %AND ZZ(2)#0) %OR (ZZ(3)#1 %AND ZZ(4)    #0) %THEN ->F1
9:
D(1) =ZZ(1)+10*(ZZ(2)+10*(ZZ(3)+10*ZZ(4)))
%CYCLE J = 0,1,5; PLIST(F,J) = D(J) ; %REPEAT
%IF F=MARKA %THEN MARKA=MARKA+1; ->1
51: ->F1 %IF ML(PC) # 'INTERVAL'; PC=PC+1
D(4) = SECTIME(STOR(ML(PC),COND)); ->F1 %C"IF COND = 'F'
->9
F1: FAULT(2,LN,'');->1
%END
```

```
      %ROUTINE INITIALIZE(   %INTEGER  Z)
      %INTEGER  J,K
%IF Z = 255 %THENSTART
%CYCLE Z=1,1,15;SBLIST(1,Z)=0;SBLIST(2,Z)=0;%REPEAT
%CYCLE J=1,1,10;INBLK(J)=-1;%REPEAT
%CYCLE J=1,1,30; VARLIST(J) =255; %REPEAT
%CYCLE Z = 1,1,64; PLIST(Z,6)=0:";PLIST(Z,7) = 0; %REPEAT
      SBLIST(1,1) = 30; SBLIST(1,2) = 20; SBLIST(1,3) = 10
SBLIST(1,4) = 60
KEY(4) = 0
MESS='';LN='';LINE='';CONTRL(5)=''
STATUS=0; %RETURN; %FINISH
      %IF Z = 0 %THEN %START
%CYCLE J = 1,1,64;%CYCLE K=0,1,7;PLIST(J,K)=0; %REPEAT ; %REPEAT
%CYCLE J=1,1,80;CONSTS(J,3) = -1; %REPEAT
%CYCLE Z = 1,1,50; SLIST(Z)=''; %REPEAT
PAR(5) = 1; PAR(6) = 1; PAR(8) = 1; PAR(9) = 1
%RETURN; %FINISH
STATUS = 3
%IF (Z=1 %AND SCANLENGTH#4) %OR  (Z=6 %AND SCANLENGR"TH #2 ) %C
%OR (2<=Z<=5 %AND SCANLENGTH # 5 ) %THEN FAULT(108,LINE,'WRONG LENGTH')
B(1)=-49;B(2)=3539;B(3)=100 ; B(4)=55;B(5)=90
      B(6) = -46; B(7) =3625; B(8) = 30; B(9) =4; B(10) =25
      B(11) = -9; B(12) = 1130; B(13) = 7.5; B(14) = 760; B(15) = 0
%IF Z = 6 %THENSTART
VARLIST(1)=2;VARLIST(2)=1
B(1)=78;B(2)=1258;%RETURN;%FINISH
VARLIST(1)=47;VARLIST(2)=2;VARLIST(3)=5;VARLIST(4)=1
%IF Z=1 %THENRETURN
%IF Z=2 %THEN VARLIST(5)=49 %ELSE VARLIST(5)=6
%IF Z = 4 %THENSTART ; B(3)=250; B(4) = 80; B(5) = 130; %FINISH
%IF Z=5 %THEN VARLIST(3)=49
      %END
      %ROUTINE CHECKSTATS(%SHORTINTEGERNAME J)
1: READLINE(LN,1)
%IF EXAMINE(LN,'GO')=1 %OR EXAMINE(LN,'BEGIN')=1%THEN MUDDLE
->END %IF LN='ENDSTATS'; ->F1 %IF J>50
    SLIST(J) = LN; J = J + 1 ; ->1
F1: FAULT(56,'','')
2:READLINE(LN,255); ->2 %UNLESS LN='ENDSTATS'
END:  SLIST(J)=LN
      %END
%ROUTINE  ADDVAR(%INTEGER J)
KEY(4)=KEY(4)+1;FAULT(114,WORD(J+120),'') %IF KEY(4)>30
VARLIST(KEY(4))=J; %END
      %REALFN S TO R(%STRINGNAME S,COND)
%INTEGER I,K,SIGN
%STRING(10) IN,RL
SIGN=1;COND='I';K=0;RL=''
%IF S->IN.('.').RL %THENSTART
COND='R';K=GETNO(RL)
%FINISHELSE IN=S
%IF IN->('-').IN %THEN SIGN=-1
%IF IN->('+').IN %THEN SIGN=+1
I=GETNO(IN)
%IF I<0 %OR K<0 %THEN->F1 %ELSERESULT=SIGN*(I+(K*(10**(-LENGTH(RL)))))
F1: COND='F'; FAULT(1,S,''); %RESULT=0
%END
```

```
%INTEGERFN GETNO(%STRINGNAME S)
%INTEGER M,L,K; K=0
%CYCLE M=1,1,LENGTH(S); L=CHARNO(S,M)-'0'; %UNLESS 0<=L<=9 %THEN->1
K=(10*K)+L ; %REPEAT
%RESULT=K
1: %RESULT=-1; %END
     %INTEGERFN SECTIME(%REAL U)
%RESULT = 60*INTPT(U) + INT(100*FRACPT(U))
     %END
     %REALFN TRUETIME(%INTEGER U)
     %RESULT = INTPT(U/60) + 0.6*FRACPT(U/60)
   %END
%ROUTINE  BIOLUNITS
%BYTEINTEGER Z,ZA,ZB,ZC
%STRING(3) SC,SD
%INTEGER J
1: READLINE (LN,1); ->9 %IF LN = 'ENDUNITS'
DECOMP(LN,ML,ZA); Z= 0
%IF ML(1)->('CH').SC %THENSTART
ZC =GETNO(SC)
->F2 %UNLESS1<=ZC<=SCANLENGTH
IDENT(ML(2),PAR(10)); PAR(10) =MEASURE(PAR(10))
->F2 %IF PAR(10)=0
VARLIST(ZC)=PAR(10); ->2
%FINISH
%IF ML(1)= 'A' %THENSTART
IDENT(ML(2),PAR(10))
PAR(10)=MEASURE(PAR(10))
%CYCLE J = 1,1,SCANLENGTH; ZC=J
%IF PAR(10)=VARLIST(J) %THEN ->2
%REPEAT
FAULT(30,ML(2),'') ; ->1; %FINISH
->F1
2:
Z=1 %IF VARLIST(ZC) = 2;Z=6 %IF VARLIST(ZC) = 5; Z=11 %IF VARLIST(ZC)= 6
->F4  %IF Z # 0  %AND ZA # 7
->1 %UNLESS 1<=Z<=11
%CYCLE J = 3,1,7
%IF STATUS = 2 %AND ML(J) = 'X' %THEN ->F5
%IF ML(J) #  'X' %THENSYMTART
B(Z+J-3)=STOR(ML(J),SC); ->F1 %COMMENT IF SD='F'
%FINISH; %REPEAT
->1
9: %IF STATUS = 2 %THENSTART
%CYCLE J = 1,1,SCANLENGTH; %IF VARLIST(J)     =255 %THEN ->F6
%REPEAT
STATUS = 3; %FINISH
->END
F4: FAULT(59,LN,'');->1
F1: FAULT(60,LN,'');->END
F2: FAULT(55,ML(2),''); ->1
F5: FAULT(58,LN,'');->1
F6: FAULT(57,RTCS(J,2),'')       ; ->END
END: %END
```

```
%INTEGERFN MEASURE(%INTEGER K)
%IF 121<=K<=153 %THENRESULT=K-120
%IF 161<=K<=193 %THENRESULT=K-160
%IF 157<=K<=160 %THENRESULT=K-110
%IF 197<=K<=200 %THENRESULT=K-150
%RESULT = 0
%END
%INTEGERFN      BSCAN(%INTEGER J)
%RESULT= 1+SCANLENGTH*((J-1)//SCANLENGTH)
%END
%INTEGERFN ESCAN(%INTEGER J)
%RESULT= BSCAN(J+SCANLENGTH) -1
%END
%SHORTINTEGERMAP FILESTATE; %RESULT = ADDR(FS(PAR(1),3)); %END
%BYTEINTEGERMAP  STATUS ; %RESULT = ADDR(KEY(2)); %R"END
      %BYTEINTEGERMAP CCC(%INTEGER V)
%INTEGER KA
KA=((V-1)//2)+1
%RESULT = ADDR(FS(KA,5)) %IF PARITY(V) = -1
%RESULT =ADDR(FS(KA,5))+1
%END
%ROUTINE       SETUP(%STRING(30) S,%SHORTINTEGERNAME  LOGFL)
%STRING(10) %ARRAY ST(1:3)
%INTEGER J; %BYTEINTEGER CC
DECOMP(S,ST,CC)
S=ST(1); LOGFL=0
%IF S->('*').S %THENSTART;LOGFL=GETNO(S);->FOUND;%FINISH
%CYCLE J =1,1,140; LOGFL=J; ->FOUND %IF NAME(J) =S; %REPEAT
NEWLINE; FAULT(3,S,''); LOGFL=0
FOUND: %IF ST(2)='BASIC' %AND(LOGFL=0 %OR LOGFL>100) %THEN FAULT %C
(101,S,'')
%RETURNIF LOGFL=60
%IF ST(2) = 'BASIC' %THENSTART
NEWLINE
PRINTSTRING('FILE '.S.' RETRIEVED - LOGICAL FILE NO IS')
WRITE(LOGFL,4); J1 = ADDR(FS(LOGFL,4))
CC=1
%CYCLE J=1,1,200;%IF CCC(J)=LOGFL %THENSTART;INBLK(CC)=J;CC=CC+1
%FINISH; %REPEAT
SCANLENGTH=BYTEINTEGER(J1);RATE=BYTEINTEGER(J1+1)
%PRINTTEXT'
SCANLENGTH = '; WRITE(BYTEINTEGER(J1),2); %PRINTTEXT' SCAN RATE = '
WRITE(BYTEINTEGER(J1 + 1),2); %FINISH
%IF LOGFL <101 %THENRETURN
%IF (101>LOGFL>120 %AND ST(2)='PRD') %OR (121>LOGFL>140 %AND %C
ST(2)='ST') %THENSTART
FAULT(3,S,'IS NOT A '.ST(2)); LOGFL=0; %FINISH
%END
%ROUTINE DECOMP(%STRING(80) LN,%STRINGARRAYNAME Z,%BYTEINTEGERNAME  NO)
%STRING(1) CH
CH = ' '
NO=0;SQUASH(LN);%IF LN='' %THENRETURN;NO=1
1:
%IF LN ->Z(NO).(CH).LN %THEN ->3 %ELSE ->4
3:  SQUASH(LN); NO = NO + 1 ; ->1
4:   Z(NO) = LN
%END
```

```
%ROUTINE DISCTRANSFER(%INTEGER K,%ROUTINE R)
%ROUTINESPEC R(%INTEGER CH,%INTEGERNAME SECT, %NAME B,E)
%INTEGER DISC,J
%IF K = 1 %THEN %START
DISC=1;R(10,DISC,BYTEINTEGER(STAD(1)),BYTEINTEGER(STAD(2)))
DISCTRANSFER(2,READDA)
DISCTRANSFER(5,READDA)
%FINISH
%IF K = 2 %THEN %START
DISC=201; R(12,DISC,FS(1,1),FS(100,5))
DISC=202;R(12,DISC,BYTEINTEGER(STAD(3)),BYTEINTEGER(STAD(8)))
%FINISH
%IF K = 3 %THEN %START
DISC=18; R(10,DISC,BYTEINTEGER(STAD(5)),BYTEINTEGER(STAD(6)))
%FINISH
%IF K = 4 %THEN %START
DISC=16; R(10,DISC,PLIST(1,3"0),PLIST(64,7))
%FINISG"H
%IF K=5 %THENSTART
DISC=7; R(10,DISC,LIB(101,1),LIB(140,2))
DISC=6;R(10,DISC,BYTEINTEGER(ADDR(NAME(101))),BYTEINTEGER(STAD(4)))
%FINISH
%IF 101<=K<=120 %THEN %START
DISC = K-100; R(11,DIA"SC,PLIST(1,0),PLIST(64,7))
%FINISH
%IF 121<=K<=140 %THEN %START
DISC=3*(K-120)+10; R(11,DIA"SC,BYTEINTEGER(STAD(5)),BYTEINTEGER   %C
(STAD(7)))
%FINISH;  %END
%ROUTINE SQUASH(%STRINGNAME  S)
%IF S=''  %OR (CHARNO(S,1) # ' ' %AND CHARNO(S,LENGTH(S))# ' ' ) %C
%THENRETURN
%IF CHARNO(S,1) = ' ' %THEN S->(' ').S
%IF CHARNO(S,LENGTH(S)) = ' ' %THEN S=FROMSTRING(S,1,LENGTH(S)-1)
SQUASH(S)
   %END
%STRINGFN  R TO S(%REAL Z , %INTEGER SIGFIGS)
%REAL X
%STRING(20)  DUM
%INTEGER  NUM,TYPE,J
X=Z; Z = |"|Z|; DUM=''
%IF -1@-7<=FRACPT(Z) <= 1@-7 %THEN TYPE = 1 %ELSE TYPE = 2
NUM = INTPT(Z); J = 1
1:  DUM = TOSTRING(FR(NUM,10)+'C').DUM ; ->3 %IF J = SIGFIGS
J=J+1;NUM=NUM//10;->1 %UNLESS NUM=0;   ->3 %IF TYPE=1
DUM = DUM.'.' ; Z = FRACPT(Z)
2:Z=10*Z;NUM=INTPT(Z);Z=FRACPT(Z)
DUM = DUM .TOSTRING(NUM + '0'); ->3 %IF J = SIGFIGS
J =  J+1; ->2
3:  DUM= '-'.DUM %IF X<C; %RESULT = DUM
%END
%INTEGERFN  EXAMINE(%STRING(100) S,%STRING(12) SUBSTR)
%STRING(100) SA
%IF S ->SA.(SUBSTR).S *"%THEN %RESULT = 1 %ELSERESULT=0
%END
```

```
      %ROUTINE TITLE(%BYTEINTEGER VAL,A)
%IF 0<=A<=1 %THEN PRINTSTRING('  '.WORD(VAL+160))
%IF A=2 %THEN PRINTSTRING('  '.WORD(109+VAL).' TRANSFORMATION')
%END
%ROUTINE  MCPT(%STRING(100) SS)
%OWNLONGREAL  XC = 0
%LONGREAL  XA
%IF KEY(3)<2 %OR STATUS=253 %THENRETURN
XA=CPUTIME;SELECTOUTPUT(9)
PRINT(XA,3,5);SPACES(3);PRINT(XA-XO,3,5);PRINTSTRING('   '.SS)
XO=XA;SELECTOUTPUT(99);%END
%ROUTINE       FAULT(%INTEGER J,%STRING(80)  AA,AB)
%IF KEY(3)>2 %THEN ->1
%PRINTTEXT'
***FAULT ';WRITE(J,3); %PRINTTEXT'***'
PRINTSTRING('    '.AA.'   '.AB); NEWLINE
MCPT('FAULT '.RTOS(J,3))
1:
%IF J>100 %OR KEY(6) =0 %THEN MUDDLE
%IF J>50 %THEN STATUS V"= 20
%END
%ROUTINE MUDDLE
%REAL X;X=SQRT(-|"|J|);%END
%INTEGERFN  FR(%INTEGER Z,DIV)
%IF Z=0 %THENRESULT=0
%RESULT=INT(Z/|Z|)*(|Z|-DIV*(|Z|//DIV))
      %END
%ROUTINE READLINE(%STRINGNAME LN, %BYTEINTEGER  TR)
%STRING(1) ONE,SNL
10:LN='';SNL=TOSTRING(NL)
1: READITEM(ONE); ONE = ' ' %IF ONE = ','
->2 %IF ONE=SNL %OR (TR=255 %AND ONE= ' ')
LN = LN.OM"NE ; ->1
2: SQUASH(LN); %IF EXAMINE(LN,'|') = 1 %OR LN='' %THEN ->10
%IF 0<TR<3 %THEN PRINTSTRING(SNL.LN)
%END
   %END    %OF    %PROGRAM
```

4: Program for analysis of the psychophysiological data

```
C       S.V.ROSENTHAL,REHC22,DEPT PSYCHIATRY,R.E.H
        DIMENSION SS(126),STCT(40),SAVE(40),SSD(40),SNN(40),
       X R(900),B(900),ITITL(10)
        REAL N1(800),N2(800),RX(2000)
        DIMENSION IFL2(40),CP(8),X(4500)
        COMMON/AL/ALS(5,50),STD(2,50)
        COMMON/CC/IFL(40),ISUB(126),IDENT(126),E(40,126),IVDNT(40),
       X ILIST(40),ISNOS(126),MM(7,30)
        NAMELIST/FUDGE/E,STD,ALS
C       IVDNT HOLDS NAMES OF VARIABLES IN E ARRAY
C       ILIST CONTAINS NAMES AS THEY ARE SELECTED FOR X ARRAY
        DO 99 K=0,6
     99 READ (5,98)  (IDENT(18*K+J),J=1,18)
     98  FORMAT(18I4)
C      INITIALIZE  ARRAYS
        DO 97 J=1,50
        ALS(5,J)=-999.0
        STD(1,J)=-999.0
   97   STD(2,J)=-999.0
        DEFINE FILE 1 (126,320,L,IASS)
        DEFINE FILE 2(580,320,L,IAST)
C        READING IN OF OPERATION CARDS
   100 NOPS=1
        WRITE(6,1013)
   101 READ(5,1010) J,(MM(I,NOPS),I=1,6),MM(7,NOPS),(ITITL(K),K=1,10)
        IF (MM(1,NOPS).EQ.99) GO TO 109
        IF(J-1) 108,102,108
   102  WRITE(6,1012) NOPS,(MM(I,NOPS),I=1,6),MM(7,NOPS),ITITL
        IF (MM(1,NOPS).GT.40.OR.MM(2,NOPS).GT.9) GO TO 108
        DO 103 I=3,6
        IF(MM(I,NOPS).GT.120) GO TO 108
   103  CONTINUE
        NOPS=NOPS+1
        GO TO 101
   108  WRITE(6,1011) NOPS,ITITL
        GO TO 101
   109  NOPS=NOPS-1
        IF(NOPS.EQ.0) GO TO 200
  1010 FORMAT(I1,2I2,4I3,A4,19X,10A4)
  1011 FORMAT(1HC,'*******INVALID OPERATION CARD****',I3,10A4)
  1012 FORMAT(1H ,7(I3,2X),A4,2X,10A4)
  1013 FORMAT(1H1,'OPERATION CARDS READ IN BEGINS')
   111 CALL NEWVAR(NOPS)
        DO 199 J=1,NOPS
   199  IVDNT(MM(1,J))=MM(7,J)
   200 READ(5,201) ICO,ICALC,IOPT,ITR,IALS,NVARS,(IFL2(I),I=1,40),
       X (ITITL(J),J=1,7)
   201 FORMAT(I1,4I2,41I1,7A4)
C        CHECK COLUMN  1
        IERR=200
        WRITE(6,205) ITITL
        IF(ICO.EQ.0) GO TO 200
        IF(ICO.NE.2) GO TO 399
C       OPERATION CARD CHECK
        IF(ICALC.EQ.97) GO TO 208
        IF(ICALC.EQ.98) GO TO 209
        IF(ICALC.EQ.99) GO TO 100
```

```
  205  FORMAT(1HO, 10A4)
       IF (ICALC.EQ.-1) STOP 1
C       CHECK FOR CHANGING LIST OF SUBJECTS
       IF (ICALC-0) 202,203,202
  203    CALL SUBJNO(ICNTS,NVARS)
       GO TO 200
  202 ICNTV=0
       DO 206 J=1,40
        IFL(J)=IFL2(J)
       IF(IFL(J).EQ.0) GO TO 206
       ICNTV=ICNTV+1
       ILIST(ICNTV)=IVCNT(J)
  206  CONTINUE
  210 GO TO (310,320,330,340,350,360,370,380,390,400,410,420),ICALC
  310  IERR=310
       IF(ICNTV.GT.4) GO TO 399
       CALL SELECT(X,N,ICNTS,ICNTV)
       DO 312 J=1,ICNTV
       CALL CHOP(X,SS,ICNTS,ICNTV,J)
       CALL RANK(SS,N2,ICNTS)
       GO TO (3120,3121,3122,3123),J
 3120 CALL TRANAR(N2,R,ICNTS)
       GO TO 312
 3121 CALL TRANAR(N2,RX,ICNTS)
       GO TO 312
 3122 CALL TRANAR(N2,B,ICNTS)
       GO TO 312
 3123 CALL TRANAR(N2,N1,ICNTS)
  312 CONTINUE
       WRITE(6,3130) (TITL(J),J=1,7)
       WRITE(6,3132) (ILIST(J),J=1,ICNTV)
       DO 314 J=1,ICNTS
       DO 3141 JA=1,ICNTV
       OP(2*JA-1)=X((JA-1)*ICNTS  +J)
       IF (JA.EQ.1) OP(2)=R(J)
       IF (JA.EQ.2) OP(4)=RX(J)
       IF (JA.EQ.3) OP(6)=B(J)
       IF (JA.EQ.4) OP(8)=N1(J)
 3141 CONTINUE
       JB=2*ICNTV
  314  WRITE(6,3131) ISNOS(J),(OP(IERR),IERR=1,JB)
       IF(IOPT-1) 200,318,200
  318 DO 3180 K=1,ICNTS
 3180 WRITE(7,3181) ISNOS(K),(OP(J),J=2,JB,2)
 3181 FORMAT(I3,2X,4E12.5)
       GO TO 200
 3130   FORMAT(1H1,20X,10A4)
 3131 FORMAT(1H ,5X,I5,5X,4(F10.4,1X,F5.1))
 3132 FORMAT(1H ,'SUBJECT NO           ',4(A4,12X))
```

```
  320 IERR=320
      IF (ICNTV.NE.2) GO TO 399
      IERR=321
      IF(IOPT.LE.0   .OR.IOPT.GT.50 ) GO TO 399
      CALL SELECT (X,N,ICNTS,ICNTV)
      JPSTR=0
      JSTR=0
      DO 321 J=1,40
      IF(IFL(J).EQ.1) JPSTR=J
      IF(IFL(J).EQ.2) JSTR=J
  321  CONTINUE
      IERR=322
      IF(JPSTR.EQ.0 .OR.JSTR.EQ.0) GO TO 399
      IF(JPSTR.GT.JSTR) GO TO 322
      JA=1
       JB=2
      GO TO 323
  322  JA=2
      JB=1
  323 CALL CHOP(X,R,ICNTS,ICNTV,JA)
      CALL CHOP(X,B,ICNTS,ICNTV,JB)
      N=ICNTS
      CALL CORPPM(R,B,N,CORR,A1,SD1,A2,SD2)
      IERR=324
      IF (CORR.EQ.99.99) GO TO 399
      ALS(1,IOPT)=A1
      ALS(2,IOPT)=SD1
      ALS(3,IOPT)=A2
      ALS(4,IOPT)=SD2
      ALS(5,IOPT)=CORR
      WRITE(6,324) A1,SD1,A2,SD2,CORR,IVONT(JPSTR),IVONT(JSTR),IOPT
      GO TO 200
  324 FORMAT(1H ,'MEAN PRE',E13.5,5X,'SD PRE = ',E13.5,5X, 'MEAN POST ='
     X ,E13.5,5X,'SD POST = ',E13.5,5X,'CORR = ',F7.3//
     X ' VARIABLES',2(2X,A4),'     ARRAY POSITION ',I2)
  350 IERR=350
  360 IERR=360
      IF(ICNTV.LT.2) GO TO 399
      IERR=361
      CALL SELECT(X,N,ICNTS,ICNTV)
C     THE USER MUST ENSURE THAT THERE ARE NO MISSING VALUSE IN THE  MAT.
      DO 369 KA=1,ICNTV
      DO 369 KB=KA,ICNTV
      IF (KA-KB) 362,361,362
  361  CORR=1
      SIG=0.0
      GO TO 368
```

```
  362  CALL CHOP(X,N1,ICNTS,ICNTV,KA)
       CALL CHOP(X,N2,ICNTS,ICNTV,KB)
       IF(KA.EQ.-1.OR.KB.EQ.-1) GO TO 399
       IF(ICALC.EQ.5) GO TO 351
       CALL SRANK(N1,N2,B,ICNTS,CORR,SIG,NDF,C)
  368  CALL LOC(KA,KB,IR,ICNTV,ICNTV,1)
       R(IR)=CORR
       RX(IR)=SIG
       GO TO 369
 351    N=ICNTS
       CALL CORPPM(N1,N2,N,CORR,A1,SD1,A2,SD2)
       CALL LOC(KA,KB,IR,ICNTV,ICNTV,1)
       R(IR)=CORR
       SAVE(KA)=A1
       SSD(KA)=SD1
       SAVE(KB)=A2
       SSD(KB)=SD2
  369  CONTINUE
       IF(ICALC.EQ.5) GO TO 354
       WRITE(6,363)
       CALL PRINMT(R,ICNTV)
       WRITE(6,364)
       CALL PRINMT(RX,ICNTV)
       GO TO 200
 354     WRITE(6,3510)
       IF(IOPT.EQ.2) GO TO 359
       CALL PRINMT(R,ICNTV)
       WRITE(6,412)
       DO 352 J=1,ICNTV
  352     WRITE(6,3512) J,ILIST(J),SAVE(J),SSD(J)
       IF(IOPT.EQ.1) CALL FACTO(R,X,STOT,SAVE,SNN,ICNTV)
       GO TO 200
  359  DO 3592 KB=1,ICNTV
       DO 3591 KA=1,ICNTV
       CALL LOC(KA,KB,IR,ICNTV,ICNTV,1)
 3591  B(KA)=R(IR)
 3592 WRITE(ITR,3593) (B(J),J=1,ICNTV)
       GO TO 200
 3593 FORMAT(10F6.3)
 3510 FORMAT(1HO,'PRODUCT MOMENT CORRELATION MATRIX')
  363  FORMAT(1HO, 'SPEARMAN RANK CORRELATION MATRIX')
  364 FORMAT(1HO,     ' SIGNIFICANCE MATRIX')
 3512 FORMAT(1H ,I4,4X,A4,3X,2E10.3)
```

```
  370  IERR=370
       IF(ICNTV.NE.1) GO TO 399
       IERR=371
       IF(IOPT.LE.0.OR.ICPT.GT.40) GO TO 399
       CALL SELECT(X,N,ICNTS,ICNTV)
       DO 371 J=1,126
       IF(ISUB(J).EQ.1) E(ICPT,J)=TRANS(X(J),ITR)
  371  CONTINUE
       GO TO 200
  380  IERR=380
       IF(ICNTV.NE.1) GO TO 399
       CALL SELECT(X,N,ICNTS,ICNTV)
       CALL TALLY(X,STOT,SAVE,SSD,SNN,ICNTS,ICNTV)
       IERR=381
       IF(IOPT.GT.50.OR.ICPT.LT.0) GO TO 399
       STD(1,IOPT)=SAVE(1)
       STD(2,IOPT)=SSD(1)
       WRITE(6,381)
       WRITE(6,3512)  IOPT,ILIST(1),SAVE(1),SSD(1)
       GO TO 200
  330  IERR=330
       IF(IOPT.GT.9) GO TO 399
       WRITE(2'1+64*(ICPT-1)) IVCNT,E
       WRITE(6,331) ICPT
       GO TO 200
  340  IERR=340
       IF(IOPT.GT.9) GO TO 399
       READ (2'1+64*(ICPT-1)) IVCNT,E
       WRITE(6,341) ICPT
       GO TO 200
  331 FORMAT(1HO,'DATA MATRIX SAVED IN POSITION ',I4)
  341 FORMAT(1HO, 'DATA MATRIX RETRIEVED FROM POSITION',I4)
  381 FORMAT(1HO,'MEAN AND SD FOR STANDARDIZED DATA'/)
  390 IAST=577
       WRITE(2'IAST)   ALS
       GO TO 200
  400 IAST=577
       READ(2'IAST) ALS
       GO TO 200
  391  CONTINUE
  410 CALL SELECT(X,N,ICNTS,ICNTV)
       CALL TALLY(X,STOT,SAVE,SSD,SNN,ICNTS,ICNTV)
       WRITE(6,412)
       DO 419 J=1,ICNTV
  419 WRITE(6,411) ILIST(J),SAVE(J),SSD(J),SNN(J)
       GO TO 200
  411 FORMAT(1H , 1X, A4, 11X, 2E12.5, 6X, F3.0)
  412 FORMAT(1HO,'VARIABLE NAME          MEAN          S.D          N')
  420 CALL SELECT(X,N,ICNTS,ICNTV)
C      NOTE THAT INPUT MATRIX IS DESTROYED
       CALL KENDL(X,R,ICNTS,ICNTV,RX)
       GO TO 200
  399 WRITE(6,3990) IERR
       GO TO 200
 3990 FORMAT(1HO, '****** ERROR NO ',I4)
  208    CALL CARDS(ICPT,ITR)
       GO TO 200
  209  READ(5,FUDGE)
       GO TO 200
       END
```

```
      SUBROUTINE NEWVAR(NOOPS)
      COMMON/AL/ALS(5,50),STD(2,50)
      COMMON/CC/IFL(40),ISUB(126),IDENT(126),E(40,126),IVDNT(40),
     X ILIST(40),ISNOS(126),MM(7,30)
      DIMENSION D(120)
  101 FORMAT(1HC,'NEWVAR ROUTINE ENTERED')
      WRITE(6,101)
      DO 100 L=1,126
      READ(1'L) (D(I),I=1,80)
      IF (L.NE.126) FIND (1'L+1)
      DO 8 I=1,40
    8 D(I+80)=E(I,L)
      DO 99 K=1,NOOPS
      IOUT=MM(1,K)
      IOPP=MM(2,K)
      IFA=MM(3,K)
      IFB=MM(4,K)
      IFC=MM(5,K)
      IFD=MM(6,K)
      X=-999
      IF(D(IFA).LT.-100) GO TO 99
      GO TO (10,20,30,40, 50,60,70,80,90),IOPP
      GO TO 99
   10  X=(D(IFA)+D(IFB))/2.0
      GO TO 99
   20    X=D(IFC)-0.5*(D(IFA)+D(IFB))
      GO TO 99
   30 X= D(IFA)-D(IFB)
      GO TO 99
   40  IF(ABS(D(IFB)).LT.0.00001.OR.D(IFB).LT.-100) GO TO 99
      X=100*D(IFA)/D(IFB)
      GO TO 99
   50  IF(ABS(D(IFB)).LT.0.00001.OR.D(IFB).LT.-100) GO TO 99
      X=ALOG10(D(IFA)/D(IFB))
      GO TO 99
   60 X=D(IFA)
      GO TO 99
   70  X=ALAB(D(IFA),D(IFB),IFC)
      GO TO 99
   80  X=STAND(D(IFA),STD(1,IFB),STD(2,IFB))
      IF(X.EQ.-999  ) GO TO 99
      X=50+10*X
      GO TO 99
   90  X=TRANS(D(IFA),IFB)
   99 E(IOUT,L)=X
  100 CONTINUE
      RETURN
      END
      SUBROUTINE  TRANAR(GIN,GOUT,N)
      DIMENSION GIN(N),GOUT(N)
      DO 1 J=1,N
    1 GOUT(J)=GIN(J)
      RETURN
      END
```

```
      SUBROUTINE  CHCP (A,X,NO,NV,IV)
      DIMENSION X(1),A(1)
      IF (IV.GT.NV) GO TO 99
      L=NO*(IV-1)
      DO 1 J=1,NO
      K=L+J
    1 X(J)=A(K)
      RETURN
   99 IV=-1
      RETURN
      END
      SUBROUTINE SELECT    (X,N,NO,NV)
      COMMON/CC/IFL(40),ISUB(126),IDENT(126),E(40,126),IVONT(40),
     X ILIST(40),ISNOS(126),MM(7,30)
      DIMENSION X(1)
      N=NO*NV
      ICS=0
      DO 29 L=1,126
      IF (ISUB(L).EQ.0) GO TO 29
      ICS=ICS+1
      ICC=0
      DO 29 I=1,40
      IF(IFL(I)) 29,29,11
   11 ICC=ICC+1
      J=ICS+(NO*ICC)-NO
      X(J)=E(I,L)
   29  CONTINUE
      WRITE(6,33) (ILIST(J),J=1,NV)
      RETURN
   33 FORMAT(1HO,'VARIABLES SELECTED ARE '/(1H ,10(1X,A4)))
      END
      REAL FUNCTION TRANS(X,KEY)
      IF( X.EQ.-999) GO TO 100
      GO TO (100,101,102,103), KEY
  100 TRANS=X
      RETURN
  101 TRANS=ALOG10(X)
      RETURN
  102 TRANS=1/X
      RETURN
  103 TRANS=SQRT(X)
      RETURN
      END
      REAL FUNCTION ALAB(PRE,POST,NO)
      COMMON/AL/ALS(5,50),STD(2,50)
      Y=ALS(5,NO)
      IF(PRE.EQ.-999.OR.POST.EQ.-999.OR.ALS(5,NO).EQ.-999) GO TO 1
      SPRE=STAND(PRE,ALS(1,NO),ALS(2,NO))
      SPOST=STAND(POST,ALS(3,NO),ALS(4,NO))
       X=(SPOST-(Y*SPRE))/SQRT(1-(Y*Y))
      ALAB=50+(10*X)
      RETURN
1        ALAB=-999
      RETURN
      END
```

```
      SUBROUTINE KENDL(X,Z,NC,NV,Y)
      DIMENSION X(1),Z(1),Y(1)
      IF (NO*NV.GT.2000) GO TO 99
      NR=0
      CALL GMTRA(X,Y,NC,NV)
C       Y MATRIX IS NV ROWS, NO COLUMNS
      CALL WTEST(Y,X,NV,NC,Z,W,CS,NDF,NR)
      WRITE(6,1) W,CS,NDF
      RETURN
   99 WRITE(6,2)
      RETURN
    1 FORMAT(1H ,//' KENDALL COEFFICIENT OF CONCORDANCE'/ ' W= ',E10.4
     X/' CHI SQUARE = ',E10.4/ ' DEGREES OF FREEDOM =',I5)
    2 FORMAT(1HO,'*****ARRAY TOO LARGE****')
      END
      SUBROUTINE SUBJNO(ICNTS,NOCRDS)
      DIMENSION  INPT(18)
      COMMON/CC/IFL(40),ISUB(126),IDENT(126),E(40,126),IVONT(40),
     X ILIST(40),ISNOS(126),MM(7,30)
C     THIS ROUTINE READS CARDS WITH SUBJECT IDENTIFIERS ON AND SELECTS
C     SUBJECTS APPRIPRIATELY .IARR CONTAINS 1 BR 0 FOR EACH OF 126 SUBS
      DO 1 J=1,126
    1 ISUB(J)=0
      ICNTS = 0
      DO 2 J=1,NOCRDS
      READ(5,90) INPT
      DO 2 I=1,18
      IF(INPT(I).EQ.0) GO TO 2
      DO 3 K=1,126
      IF (IDENT(K)-INPT(I)) 3,5,3
    5 ISUB(K)=1
      ICNTS=ICNTS+1
      GO TO 2
    3 CONTINUE
      WRITE(6,91) J,I,INPT(I)
    2 CONTINUE
      I=1
      DO 4 J=1,126
      IF(ISUB(J).NE.1) GO TO 4
      ISNOS(I)=IDENT(J)
      I=I+1
    4 CONTINUE
      WRITE(6,92) ICNTS,(ISNOS(J),J=1,ICNTS)
      RETURN
   90  FORMAT(18I4)
   91 FORMAT(1H ,'INVALID IDENT ON CARD ',I4,'POSITION',I4,
     X 'VALUE',I4)
   92 FORMAT(1H ,I4,' SUBJECTS IN GROUP'/(1H ,30I4))
      END
      REAL FUNCTION STAND(X,M,SD)
        REAL*4 X,M,SD
      IF(X.LT.-100.OR.M.LT.-100) GO TO 101
      STAND=(X-M)/SD
      RETURN
  101 STAND=-999
      RETURN
      END
```

```
      SUBROUTINE CORPRM (X,Y,N,R,AV1,SD1,AV2,SD2)
      DIMENSION  X(1),Y(1)
      REAL*8 SX,SY,SXY,SX2,XY2,NUM,DEN,DN
      DN=0.0
      SX=0.0
      SY=0.0
      SXY=0.0
      SX2=0.0
      SY2=0.0
      DO100 I=1,N
      IF(X(I).EQ.-999.OR.Y(I).EQ.-999) GO TO 100
      SX=SX+X(I)
      SY=SY+Y(I)
      SXY=SXY+X(I)*Y(I)
      SX2=SX2+X(I)*X(I)
      SY2=SY2+Y(I)*Y(I)
      DN=DN+1.0
  100 CONTINUE
      IF(DN.LE.2.0) GO TO 101
      NUM=DN*SXY-SX*SY
      DEN=DSQRT((DN*SX2-SX*SX)*(DN*SY2-SY*SY))
      AV1=SX/DN
      AV2=SY/DN
      R=SNGL(NUM/DEN)
      SD1=SNGL(DSQRT((SX2-((SX**2)/DN))/(DN-1.0)))
      SD2=SNGL(DSQRT((SY2-((SY**2)/DN))/(DN-1.0)))
      RETURN
  101 R=99.99
      RETURN
      END
      SUBROUTINE TALLY(A,TOT,AVER,SD,SNN,NO,NV)
      DIMENSION A(1),TOT(1),AVER(1),SD(1),SNN(1)
      DO 1 K=1,NV
      TOT(K)=0.0
      AVER(K)=0.0
      SD(K)=0.0
    1 SNN(K)=0
      DO 6 J=1,NO
      IJ=J-NO
      DO 6 I=1,NV
      IJ=IJ+NO
      IF(A(IJ).EQ.-999) GO TO 6
      TOT(I)=TOT(I)+A(IJ)
      SD(I)=SD(I)+A(IJ)*A(IJ)
      SNN(I)=SNN(I)+1.0
    6 CONTINUE
      DO 8 I=1,NV
        AVER(I)=TOT(I)/SNN(I)
      SD(I)=SD(I)-(TOT(I)*TOT(I)/SNN(I))
    8 SD(I)=SQRT(SD(I)/(SNN(I)-1))
      RETURN
      END
```

```
      SUBROUTINE PRINMT(X,NCLT)
      DIMENSION RCUT(40),X(1)
      ICOUNT=1
  107 NCOUNT=ICOUNT+19
      IF(NCOUNT.GT.NOUT) NCOUNT=NOUT
      WRITE(6,8) (I,I=ICOUNT,NCOUNT)
      DO 108 I=1,NOUT
      DO 109 J=ICOUNT,NCOUNT
      CALL LOC(I,J,IR,NOUT,NOUT,1)
  109 ROUT(J)=X(IR)
  108 WRITE(6,9)I,(ROUT(J),J=ICOUNT,NCOUNT  )
      ICOUNT=ICOUNT+20
      IF(NCOUNT.NE.NOUT) GO TO 119
      RETURN
  119 WRITE(6,120)
      GO TO 107
    8 FORMAT (1HO,6X,20I6)
    9 FORMAT(1HO,I3,3X,20F6.2)
  120 FORMAT(1HO, 'CONTINUATION OF MATRIX')
      RETURN
      END
      SUBROUTINE  CARDS(NV,ISTR)
      COMMON/CC/IFL(40),ISUB(126),IDENT(126),E(40,126),IVDNT(40),
     X ILIST(40),ISNOS(126),MM(7,30)
      DIMENSION  IPOS(20),IFMT(20),RL(20)
C      FIRST CARD HAS FIXED FORMAT GIVING N NOS INDICATING POSITION IN A
      READ(5,1) (IPOS(J),J=1,NV)
      READ(5,2) IFL
      DO 9 J=1,NV
      DO 10 K=1,126
   10 E(IPOS(J),K)=-999.0
    9 IVDNT(IPOS(J))=IFL(J)
      READ(ISTR,2) IFMT
    1 FORMAT(20I4)
    2  FORMAT(20A4)
    3  READ(ISTR,IFMT) ID,(RL(J),J=1,NV)
      WRITE(6,7)   ID,(RL(J),J=1,NV)
      IF(ID.EQ.0) GO TO 99
      DO 4 J=1,126
      IF (IDENT(J)-ID)   4,5,4
    5 DO 6 K=1,NV
    6 E(IPOS(K),J)=RL(K)
      GO TO 3
    4  CONTINUE
      WRITE(6,8) ID
      GO TO 3
    7 FORMAT (1H , I4, 2X,20F6.2)
    8  FORMAT(1H ,'INVALID IDENT',I5)
99      RETURN
      END
```

```
      SUBROUTINE FACTC(CR,WX,A,B,S,NV)
      DIMENSION CR(1),WX(1),A(1),B(1),S(1),TV(51)
      MV=0
      CON=1.0
      CALL EIGEN(CR,WX,NV,MV)
C     CR IS CORR MATRIX
      CALL TRACE(NV,CR,CCN,NF,A)
      DO 130 I=1,NF
      L= I+(I*I-I)/2
  130   S(I)=CR(L)
      WRITE(6,6) (S(J),J=1,NF)
      WRITE(6,7) (A(J),J=1,NF)
      WRITE(6,8)
      L=0
      DO 150 J=1,NF
      DO 140 I=1,NV
      L=L+1
  140 A(I)=WX(L)
  150  WRITE(6,9) J,(A(I),I=1,NV)
      CALL LOAD(NV,NF,CR,WX)
      WRITE(6,10) NF
      DO 180 I=1,NV
      DO 170 J=1,NF
      L= NV*(J-1)+I
  170 A(J)=WX(L)
  180 WRITE(6,11) I,(A(J),J=1,NF)
      RETURN
    6 FORMAT(1HO,'EIGENVALUES'/(10F12.5))
    7 FORMAT(1HO,'CUMULATIVE PERCENTAGE OF EIGENVALUES'/(10F12.5))
    8 FORMAT(1HO,'EIGENVECTORS')
    9 FORMAT(1HO,'VECTOR',I3/(10F12.5))
   10 FORMAT(1HO/' FACTOR MATRIX (', I3,' FACTORS)')
   11 FORMAT(1HO,'VARIABLE ' I3/(10F12.5))
      END
```

Appendix 3

The Comparability of Data Collected by Hand and from the Data Logger

Several tests were carried out to ensure that the two methods of collecting psychophysiological data gave identical results, and that data collected by one method could be mixed for the purpose of analysis with data collected by the other.

Skin Conductance

The compatibility of the two systems was tested by reading the voltage corresponding to various fixed resistances from both recording instruments. The results are shown in Figure A3.1. It can be seen that the two methods are equivalent.

Respiration Rate

A pulse generator was used to generate signals to stimulate breathing and the resulting respiration rates were logged by hand and by the data logger. Figure A3.2 shows the relationship between the two methods of data collection. It will be seen that they are equivalent. It was not possible to test the comparability of heart rate measurements, but since the same type of ratemeter was used there is no reason to suspect that the results would be any different.

Ventilation Volume

It was not possible to test the equivalence of the two methods of measurement. This was because the two methods could not be used simultaneously for recording data. When the data logger was used, the output from the electronic integrator was reset to zero each time the integral reached a fixed level, rather than after a fixed time.

The computer program was given this level as a parameter and analysed the ventilation readings as follows:

If -

L = voltage corresponding to 'zero' integral

H = voltage corresponding to level at which integrator reset

V = volume of air (litres) equal to L - H volts

M = most recent signal from the integrator circuit (volts)

R = previous signal from integrator circuit (volts)

S = time between successive signals (in seconds)

$x = R - M$ if $M > R$

$x = M + (H - L) - R$ if $M < R$ (i.e., if the integrator "reset" between the last two readings)

$\therefore$ volume of air $y = \frac{x}{(H - L)} \times V$

in time S

$\therefore$ equivalent minute volume $= \frac{y \times 60}{s}$ litres.

When the polygraph was being used to collect data, the integrator was reset after a fixed time interval of 30 seconds. The height of the polygraph tracing was measured and the corresponding minute volume was computed using the calibration tables.

Thus it is not easy to compare the two methods, but since the parameter V above was estimated by measuring the equivalent polygraph height of (H - L) and using the same calibration tables, the two methods of data analysis should be equivalent.

Respiration Rate and "Breath Counting"

Over a period of several weeks when the respiration ratemeter was out of action, estimates of respiration rate were obtained by counting the number of breaths every minute from the polygraph record of ventilation volume. Thus at different times, three methods of assessing respiration rate were used in this research, breath counting, hand-logged respiration ratemeter readings and data-logged readings from the ratemeter.

To ensure that all three methods of measurement were equivalent, a detailed comparison of these methods was made. For 18 subjects in the study, an estimate of the mean respiration rate per minute for each of the 21 minutes of the session was made using three separate methods:

a) the number of breaths counted in one minute

b) the mean of the data logger readings for each minute

c) the mean of the two hand-logged respiration rate readings from the ratemeter in each minute.

The data matrix thus obtained was subjected to an analysis of variance. The results are shown in Table A3.1. It can be seen that there is no difference among the methods of assessing respiration rate. Thus they can be used interchangeably with a considerable degree of confidence to measure mean rates over a period of a minute or more.

TABLE A3.1

COMPARISON OF RESPIRATION RATE MEASUREMENT TECHNIQUES

Analysis of Variance

Source of Variance	df	SS	MS	F
Between Ss	17	20646.1	1214.5	
Within Ss	1116	284663.8	255.1	
Methods	2	48.2	24.1	<1
Subjects x Methods	34	49449.2	1455.9	
Times	20	674.5	33.7	1.64 ($p<0.05$)
Subjects x Times	340	7050.1	20.7	
Methods x Times	40	89.4	2.2	<1
Subjects x Methods x Times	680	227302.4	334.3	

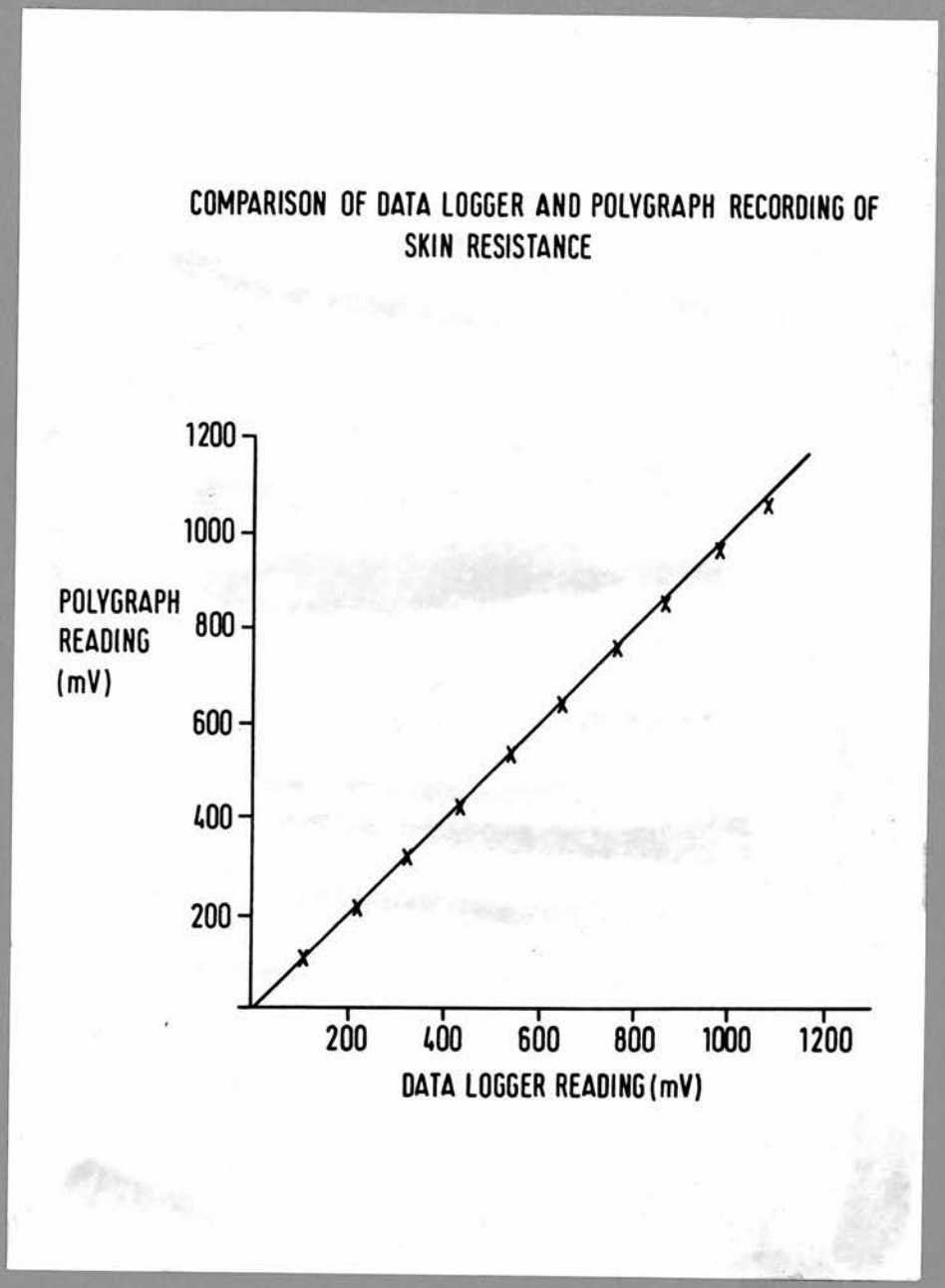

Figure A3.1

Comparison of polygraph and data logger measurements of skin resistance.

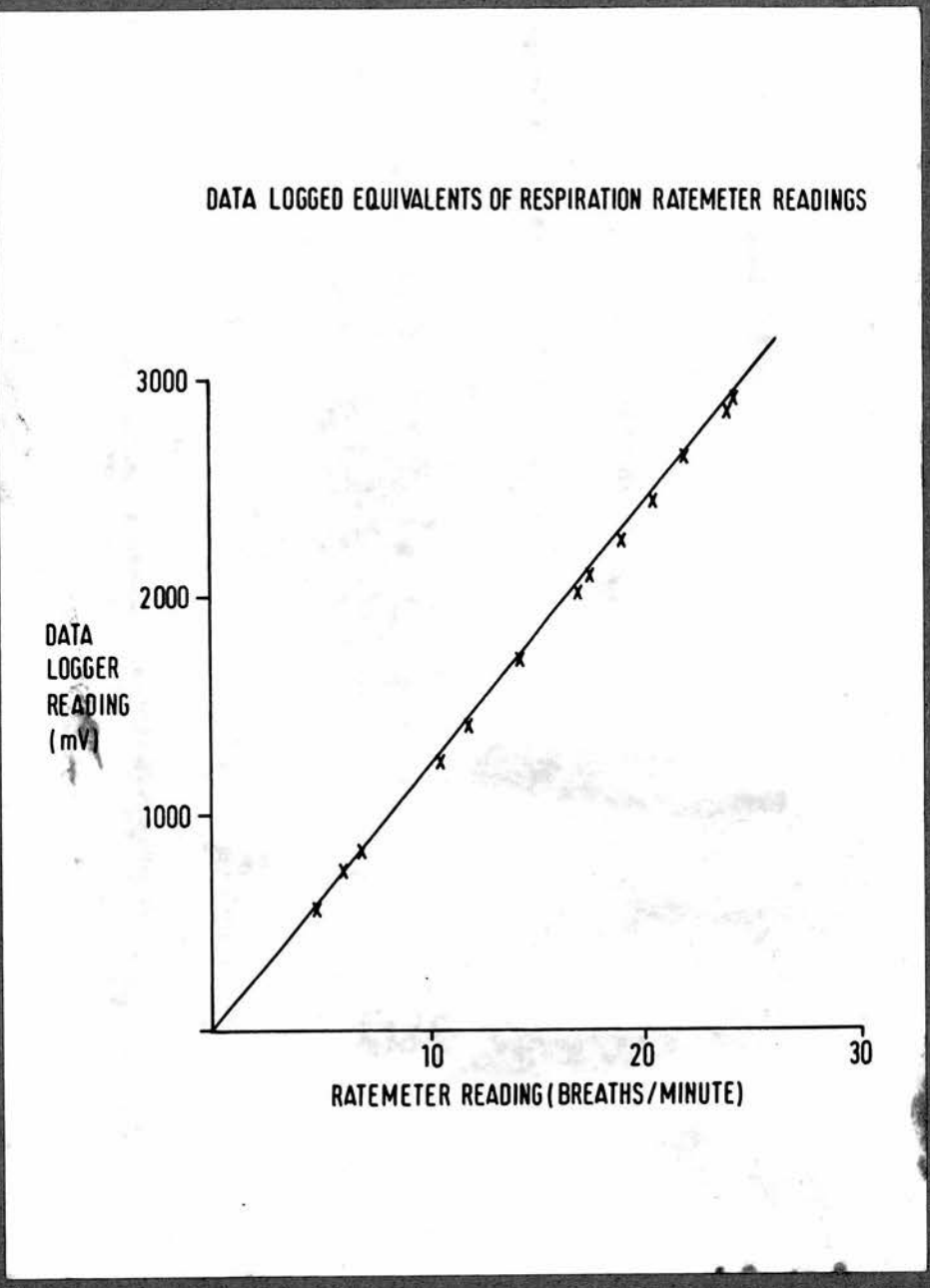

Figure A3.2

Comparison of two methods of measuring respiration rate.

APPENDIX 4

Correlation and factor matrices

In the tables in this appendix the variables are numbered according to the following key:

Identifying number	Variable
1	Skin conductance basal level
2	Skin conductance response
3	Heart rate basal level
4	Heart rate response
5	Respiration rate basal level
6	Respiration rate response
7	Ventilation volume basal level
8	Ventilation volume response
9	TMAS
10	EPI Neuroticism scale
11	EPI Extraversion scale
12	HDHQ Total Hostility
13	HDHQ Direction of hostility
14	16 PF Factor A
15	" " B
16	" " C
17	" " E
18	" " F
19	" " G
20	" " H
21	" " I
22	" " L
23	" " M
24	" " N
25	" " O
26	" " Q_1
27	" " Q_2
28	" " Q_3
29	" " Q_4

Asthmatic group's correlation matrix

1	100	-13	0	1	0	-2	4	6	-8	-1	0	6	10	4	-23	9	21	12	-6	2	-3	7	12	22	3	0	-6	-7	0
2	-13	100	0	9	2	30	-11	2	5	4	-10	-1	3	-11	0	14	-10	-9	-3	3	-5	0	-2	-13	0	-16	-3	11	3
3	0	0	100	-11	13	-1	19	12	17	4	-8	-16	24	-18	9	4	-8	-8	10	-5	0	-14	0	-1	2	-5	-9	3	-11
4	1	9	-11	100	16	20	-2	46	6	14	10	1	-9	0	-6	-15	2	3	-13	5	-12	-17	9	2	6	8	-18	13	2
5	0	2	13	16	100	-19	38	15	-5	-3	-8	-17	-4	-2	-12	6	-7	-25	27	10	14	-38	-20	-4	-12	6	-1	13	-12
6	-2	30	-1	20	-19	100	-11	22	29	22	-5	23	13	-10	4	-23	-4	-7	-17	-25	-2	27	7	7	20	-6	-4	-11	27
7	4	-11	19	-2	38	-11	100	-6	13	0	9	-15	0	-3	0	3	4	-7	14	4	14	-21	-12	-22	2	-7	7	-3	6
8	6	2	12	46	15	22	-6	100	17	21	9	20	-19	7	3	-9	0	3	-12	1	-10	7	9	10	13	14	3	-9	-2
9	-8	5	17	6	-5	29	13	17	100	76	-22	58	49	-10	5	-59	-1	-20	10	-53	26	33	11	-11	62	-10	6	-37	43
10	-1	4	4	14	-3	22	0	21	76	100	-19	60	46	-4	0	-42	0	-12	-2	-51	30	34	12	2	49	-3	11	-27	30
11	0	-10	-8	10	-8	-5	9	9	-22	-19	100	8	-53	41	1	9	31	50	-15	55	-22	14	-5	-4	-27	11	-37	-3	4
12	6	-1	-16	1	-17	23	-15	20	58	60	8	100	18	3	-10	-40	18	22	-13	-31	-3	54	7	0	37	-11	-17	-28	33
13	10	3	24	-9	-4	13	0	-19	49	46	-53	18	100	-34	-12	-35	-9	-34	10	-69	17	3	28	-6	32	-22	10	-23	13
14	4	-11	-18	0	-2	-10	-3	7	-10	-4	41	3	-34	100	-1	18	28	32	-11	30	11	6	-15	21	-6	16	-13	7	8
15	-23	0	9	-6	-12	4	0	3	5	0	1	-10	-12	-1	100	8	-6	3	19	11	17	3	5	-1	3	27	7	-6	6
16	9	14	4	-15	6	-23	3	-9	-59	-42	9	-40	-35	18	8	100	-17	3	2	31	-3	-30	-19	3	-59	-6	8	32	-48
17	21	-10	-8	2	-7	-4	4	0	-1	0	31	18	-9	28	-6	-17	100	53	-29	20	0	28	21	20	-7	-11	-23	-18	9
18	12	-9	-8	3	-25	-7	-7	3	-20	-12	50	22	-34	32	3	3	53	100	-31	33	-10	33	17	8	-9	5	-30	-14	4
19	-6	-3	10	-13	27	-17	14	-12	10	-2	-15	-13	10	-11	19	2	-29	-31	100	9	4	-12	-23	-16	0	13	12	3	-14
20	2	3	-5	5	10	-25	4	1	-53	-51	55	-31	-69	30	11	31	20	33	9	100	-31	-13	-12	8	-49	20	-32	16	-25
21	-3	-5	0	-12	14	-2	14	-10	26	30	-22	-3	17	11	17	-3	0	-10	4	-31	100	-9	17	17	18	10	20	-13	21
22	7	0	-14	-17	-38	27	-21	7	33	34	14	54	3	6	3	-30	28	33	-12	-13	-9	100	9	-6	36	-17	-13	-25	30
23	12	-2	0	9	-20	7	-12	9	11	12	-5	7	28	-15	5	-19	21	17	-23	-12	17	9	100	13	11	9	4	-28	9
24	22	-13	-1	2	-4	7	-22	10	-11	2	-4	0	-6	21	-1	3	20	8	-16	8	17	-6	13	100	-2	14	11	10	8
25	3	0	2	6	-12	20	2	13	62	49	-27	37	32	-6	3	-59	-7	-9	0	-49	18	36	11	-2	100	-5	8	-37	53
26	0	-16	-5	8	6	-6	-7	14	-10	-3	11	-11	-22	16	27	-6	-11	5	13	20	10	-17	9	14	-5	100	3	13	-16
27	-6	-3	-9	-18	-1	-4	7	3	6	11	-37	-17	10	-13	7	8	-23	-30	12	-32	20	-13	4	11	8	3	100	0	-6
28	-7	11	3	13	13	-11	-3	-9	-37	-27	-3	-28	-23	7	-6	32	-18	-14	3	16	-13	-25	-28	10	-37	13	0	100	-21
29	0	3	-11	2	-12	27	6	-2	43	30	4	33	13	8	6	-48	9	4	-14	-29	21	30	9	8	53	-16	-6	-21	100

Non-asthmatic group's correlation matrix

1	100	7	-5	26	5	15	-25	26	-25	-23	14	-16	-5	7	31	21	-20	5	-16	2	-9	0	-14	8	-29	-2	24	3	-17
2	7	100	-11	31	9	9	14	12	-10	-6	17	-22	-6	29	-2	30	-22	7	37	26	7	-11	-13	0	-32	5	20	8	4
3	-5	-11	100	0	10	12	33	21	44	52	19	41	43	0	-19	-36	-17	-7	-18	-26	-14	10	22	-32	25	-17	-3	-36	21
4	26	31	0	100	16	8	-11	47	-1	0	16	-7	0	11	0	3	8	0	-26	19	-1	-22	25	33	-15	19	16	6	-11
5	5	9	10	16	100	-68	65	-21	31	28	-6	21	12	26	-27	-23	-19	1	-20	-12	32	17	1	23	19	-34	-2	-14	25
6	15	9	12	8	-68	100	-35	48	-29	-25	16	-20	-3	6	9	26	2	-16	15	17	-44	-35	12	-25	-31	25	-2	2	-23
7	-25	14	33	-11	65	-35	100	-12	30	26	6	27	10	19	-31	-19	-13	2	-3	-9	12	8	11	0	14	-14	4	-22	26
8	26	12	21	47	-21	48	-12	100	3	2	38	1	5	16	2	12	-7	-2	-3	13	-20	-26	17	0	-8	13	-3	-4	-7
9	-25	-10	44	-1	31	-29	30	3	100	93	-1	85	45	13	-34	-77	13	-1	-25	-59	12	57	18	21	77	-27	-11	-56	70
10	-23	-6	52	0	28	-25	26	2	93	100	0	84	46	6	-42	-76	8	9	-22	-56	13	59	15	12	72	-28	-14	-52	67
11	14	17	19	16	-6	16	6	38	-1	0	100	8	-26	25	3	-8	10	40	4	52	-22	6	0	-13	-14	-2	-11	-13	24
12	-16	-22	41	-7	21	-20	27	1	85	84	8	100	15	-3	-38	-81	19	14	-35	-57	8	64	11	11	76	-31	-4	-54	67
13	-5	-6	43	0	12	-3	10	5	45	46	-26	15	100	22	-10	-27	-28	-29	-9	-37	13	-1	21	14	22	-6	-35	-24	0
14	7	29	0	11	26	6	19	16	13	6	25	-3	22	100	-12	9	-15	-13	33	25	-5	-8	-7	4	-7	10	-36	-11	5
15	31	-2	-19	0	-27	9	-31	2	-34	-42	3	-38	-10	-12	100	33	6	6	-9	16	-17	-16	-11	18	-20	17	9	8	-41
16	21	30	-36	3	-23	26	-19	12	-77	-76	-8	-81	-27	9	33	100	-29	-17	39	53	-9	-69	-5	-28	-73	23	7	47	-65
17	-20	-22	-17	8	-19	2	-13	-7	13	8	10	19	-28	-15	6	-29	100	13	-6	14	-22	35	-5	20	9	40	0	5	24
18	5	7	-7	0	1	-16	2	-2	-1	9	40	14	-29	-13	6	-17	13	100	-22	17	-3	37	-18	-7	0	-10	-12	-17	25
19	-16	37	-18	-26	-20	15	-3	-3	-25	-22	4	-35	-9	33	-9	39	-6	-22	100	31	0	-15	-18	-24	-26	29	-3	17	-7
20	2	26	-26	19	-12	17	-9	13	-59	-56	52	-57	-37	25	16	53	14	17	31	100	-34	-38	-8	-19	-73	37	-8	42	-37
21	-9	7	-14	-1	32	-44	12	-20	12	13	-22	8	13	-5	-17	-9	-22	-3	0	-34	100	-5	2	24	25	-37	-21	-1	20
22	0	-11	10	-22	17	-35	8	-26	57	59	6	64	-1	-8	-16	-69	35	37	-15	-38	-5	100	-19	14	57	-15	3	-35	63
23	-14	-13	22	25	1	12	11	17	18	15	0	11	21	-7	-11	-5	-5	-18	-18	-8	2	-19	100	29	2	17	-6	-24	-4
24	8	0	-32	33	23	-25	0	0	21	12	-13	11	14	4	18	-28	20	-7	-24	-19	24	14	29	100	21	18	5	5	7
25	-29	-32	25	-15	19	-31	14	-8	77	72	-14	76	22	-7	-20	-73	9	0	-26	-73	25	57	2	21	100	-35	0	-54	66
26	-2	5	-17	19	-34	25	-14	13	-27	-28	-2	-31	-6	10	17	23	40	-10	29	37	-37	-15	17	18	-35	100	15	27	-38
27	24	20	-3	16	-2	-2	4	-3	-11	-14	-11	-4	-35	-36	9	7	0	-12	-3	-8	-21	3	-6	5	0	15	100	1	2
28	3	8	-36	6	-14	2	-22	-4	-56	-52	-13	-54	-24	-11	8	47	5	-17	17	42	-1	-35	-24	5	-54	27	1	100	-55
29	-17	4	21	-11	25	-23	26	-7	70	67	24	67	0	5	-41	-65	24	25	-7	-37	20	63	-4	7	66	-38	2	-55	100

FACTOR MATRIX BEFORE ROTATION

VARIABLE	FACTOR 1	2	3	4	5	6
1	-0.00205	0.14176	-0.09296	0.06455	0.17975	-0.31218
2	0.03535	-0.07695	0.05194	-0.33182	-0.11261	0.04913
3	0.04441	-0.21742	0.08150	0.08507	-0.09471	-0.14102
4	0.02857	0.14236	0.59220	-0.35062	0.19276	-0.20959
5	-0.16502	-0.33664	0.47987	0.23842	-0.06587	-0.29794
6	0.36023	0.13548	0.10406	-0.37181	0.03386	0.09441
7	-0.03072	-0.18912	0.23255	0.42676	-0.25097	-0.22812
8	0.11413	0.19890	0.52628	-0.21551	0.20138	-0.01524
9	0.87869	0.00173	0.25344	0.15405	-0.16745	0.06569
10	0.75346	0.05289	0.20477	0.06612	0.06261	0.03237
11	-0.37358	0.62841	0.18378	0.13606	-0.22202	0.03177
12	0.58150	0.43882	0.04057	-0.06759	-0.13763	0.00553
13	0.65188	-0.38054	-0.26307	-0.00087	0.02607	-0.32844
14	-0.22586	0.40171	0.10366	0.24534	0.12151	0.12171
15	-0.01984	-0.02463	0.07677	0.15335	0.05114	0.44677
16	-0.63125	-0.17834	-0.13836	-0.04632	0.05030	0.04199
17	-0.01753	0.59895	-0.12075	0.25009	0.09268	-0.33350
18	-0.18477	0.73479	-0.12560	0.12891	0.02634	-0.06326
19	-0.04236	-0.39836	0.14104	0.22079	-0.23878	0.18740
20	-0.74216	0.33135	0.18842	0.08871	-0.13559	0.07042
21	0.26015	-0.17602	0.01306	0.45154	0.33127	0.11289
22	0.42780	0.53770	-0.22395	-0.07447	-0.21737	0.18461
23	0.23515	0.15946	-0.13958	0.01092	0.34253	-0.12764
24	-0.04220	0.15046	-0.02838	0.06337	0.55135	-0.00935
25	0.72169	0.05995	0.09087	0.08210	-0.00973	0.12209
26	-0.17559	0.01932	0.25687	0.14358	0.29348	0.29784
27	0.12100	-0.38528	-0.09394	0.07598	0.26370	0.19050
28	-0.40964	-0.20507	0.09034	-0.19770	0.05833	0.03157
29	0.49865	0.24534	0.03171	0.11531	-0.02932	0.08611

Unrotated factor matrix : asthmatics

ROTATED FACTOR MATRIX

VARIABLE	FACTOR 1	2	3	4	5	6
1	-0.01262	0.04739	0.00119	0.03760	-0.08869	-0.38857
2	-0.02736	-0.11592	0.16993	-0.14113	-0.17745	0.20148
3	0.00480	-0.13677	-0.00943	0.25828	-0.06993	0.03952
4	0.01841	0.06206	0.74726	0.05678	-0.05799	-0.08145
5	-0.16363	-0.05047	0.23024	0.65971	0.03647	0.04361
6	0.30912	-0.11470	0.30307	-0.30068	-0.10580	0.07126
7	0.04776	0.07163	-0.10211	0.60573	-0.01337	0.05493
8	0.14663	0.10215	0.61024	-0.01095	0.10652	-0.03148
9	0.89024	-0.21452	0.09956	0.16648	0.02324	0.12529
10	0.72843	-0.22141	0.17647	0.03696	0.08638	-0.04655
11	-0.06313	0.79287	0.05833	-0.01212	-0.02107	0.02093
12	0.67459	0.15079	0.09999	-0.19657	-0.16324	-0.05296
13	0.41573	-0.64426	-0.16711	0.12616	-0.26205	-0.21703
14	-0.03442	0.46895	0.00352	-0.03193	0.27225	-0.12904
15	0.04168	0.04900	-0.07051	-0.07747	0.39800	0.24312
16	-0.65637	0.05983	-0.12179	-0.01508	0.05052	0.06206
17	0.15669	0.48876	-0.09075	-0.00925	-0.10018	-0.52362
18	0.05479	0.66679	-0.07882	-0.23264	-0.03015	-0.31984
19	-0.04421	-0.14517	-0.14215	0.32952	0.14481	0.38532
20	-0.50697	0.66728	0.05806	0.06099	0.07329	0.11205
21	0.22745	-0.21942	-0.15768	0.19454	0.47472	-0.19301
22	0.56136	0.27236	-0.14636	-0.41446	-0.16139	0.02503
23	0.18054	-0.09349	0.02491	-0.16256	0.06695	-0.40134
24	-0.07583	-0.00834	0.11396	-0.16413	0.32829	-0.42402
25	0.71125	-0.19540	0.04576	-0.03017	0.08235	0.02410
26	-0.12028	0.10251	0.16191	0.00087	0.48988	0.03832
27	-0.02986	-0.42470	-0.10237	-0.00886	0.30237	0.01614
28	-0.46901	-0.04871	0.14712	0.00021	0.01847	0.13104
29	0.56461	0.04534	-0.00807	-0.07454	0.05180	-0.04758

Varimax rotation : asthmatics

ROTATED FACTOR MATRIX

VARIABLE	FACTOR 1	2	3	4	5	6
1	-0.03663	0.05233	0.01374	0.04946	0.36494	-0.18898
2	-0.03012	-0.12653	0.14131	-0.15203	-0.20351	-0.11775
3	0.00905	-0.11179	0.00860	0.25349	-0.04469	-0.09004
4	0.04534	0.07015	0.76311	0.10215	0.09728	-0.05810
5	-0.12486	-0.00242	0.29349	0.67390	-0.02797	-0.01156
6	0.30616	-0.11917	0.27364	-0.28554	-0.07513	-0.04688
7	0.08065	0.13613	-0.04346	0.62576	-0.09452	-0.03476
8	0.18060	0.10854	0.62663	0.03635	0.05789	0.12918
9	0.91850	-0.13687	0.12616	0.21291	-0.16390	0.08082
10	0.74185	-0.17222	0.19579	0.07584	0.04022	0.09646
11	-0.02095	0.80608	0.07360	0.05078	-0.11314	0.06338
12	0.68034	0.19204	0.09298	-0.14065	-0.03617	-0.09822
13	0.36507	-0.60631	-0.16815	0.09999	0.20234	-0.36450
14	-0.00740	0.46273	0.02597	0.00587	0.12469	0.27759
15	0.07207	0.03189	-0.06767	-0.08136	-0.18005	0.46353
16	-0.66634	0.00903	-0.13550	-0.05971	-0.01464	0.02859
17	0.14481	0.51207	-0.06672	0.04648	0.42732	-0.17965
18	0.05558	0.66352	-0.07756	-0.17863	0.23117	-0.02885
19	-0.01178	-0.12325	-0.12120	0.30964	-0.35425	0.19906
20	-0.46731	0.64801	0.07031	0.08541	-0.13941	0.13334
21	0.23413	-0.20795	-0.11554	0.19256	0.27333	0.38404
22	0.55617	0.28801	-0.17912	-0.37535	-0.12480	-0.05999
23	0.15087	-0.10268	0.02536	-0.15422	0.41587	-0.02793
24	-0.09255	-0.04562	0.12397	-0.16071	0.49997	0.21443
25	0.72167	-0.15243	0.05500	0.00019	-0.03709	0.11500
26	-0.08637	0.07655	0.18323	0.00832	0.05578	0.49260
27	-0.04215	-0.44908	-0.10275	-0.05119	0.08811	0.25172
28	-0.46701	-0.08550	0.13556	-0.02931	-0.07745	0.02173
29	0.57453	0.07949	-0.00007	-0.03628	0.00951	0.08319

Oblimin rotation : asthmatics

FACTOR MATRIX BEFORE ROTATION

VARIABLE	FACTOR 1	2	3	4	5	6
1	-0.22986	0.11448	-0.05933	0.00875	0.26281	-0.43416
2	-0.20922	0.04907	-0.27760	0.36951	0.06571	0.05967
3	0.42019	0.30855	-0.39060	-0.13678	-0.23781	-0.15098
4	-0.11663	0.29854	-0.27664	0.04326	0.67664	-0.08178
5	0.40791	-0.45548	-0.35461	0.49454	0.32647	-0.05857
6	-0.39853	0.59797	-0.12820	-0.36348	-0.22563	-0.02090
7	0.34984	-0.18204	-0.31647	0.35253	-0.03909	0.09303
8	-0.13159	0.59015	-0.35419	-0.10204	0.17445	-0.15298
9	0.93059	0.15771	-0.14624	-0.06085	0.04691	0.18333
10	0.90692	0.18542	-0.15135	-0.03055	-0.02603	0.11977
11	-0.04520	0.61675	-0.04411	0.53374	-0.01716	-0.16303
12	0.89023	0.22921	0.08416	-0.01373	-0.01567	-0.03187
13	0.34055	-0.06068	-0.49959	-0.36084	-0.01256	0.15135
14	-0.01999	0.12959	-0.45898	0.31562	-0.01204	0.30357
15	-0.37747	0.02580	0.21456	-0.15315	0.17865	-0.19412
16	-0.86703	-0.16154	-0.20098	0.03791	-0.10184	-0.05093
17	0.09460	0.31937	0.60309	0.03723	0.20884	0.38948
18	0.11176	0.19679	0.32424	0.41863	0.01648	-0.27510
19	-0.33992	-0.03234	-0.11463	0.20253	-0.38284	0.45752
20	-0.68994	0.27790	-0.00757	0.50225	0.03047	0.18367
21	0.23877	-0.48502	-0.13125	0.05912	0.09488	-0.03338
22	0.66045	0.09639	0.45871	0.20471	-0.01475	0.01803
23	0.11238	0.13817	-0.27407	-0.27537	0.23532	0.11227
24	0.18527	-0.13115	0.07319	-0.11558	0.74905	0.21277
25	0.84563	-0.04454	0.14490	-0.17193	-0.02455	-0.00061
26	-0.41734	0.28100	0.12424	-0.13972	0.23330	0.52643
27	-0.08571	-0.01289	0.16828	-0.02712	0.15683	-0.15652
28	-0.58321	-0.22430	0.11690	0.01004	0.10616	0.14048
29	0.75439	0.16107	0.13523	0.32098	-0.09614	0.02284

Unrotated factor matrix : non-asthmatics

ROTATED FACTOR MATRIX

VARIABLE	FACTOR 1	2	3	4	5	6
1	0.21188	0.05451	-0.16298	-0.15707	-0.43908	-0.18763
2	0.20610	-0.06792	-0.12180	0.42738	-0.14538	-0.04395
3	-0.48749	0.24279	0.19132	0.16633	-0.08525	-0.39276
4	0.07340	0.00361	0.05198	0.13725	-0.76805	0.16974
5	-0.20367	-0.79881	-0.03048	0.32193	-0.21293	-0.15715
6	0.18349	0.80506	0.13427	0.03080	-0.10201	-0.07161
7	-0.26016	-0.37495	0.01387	0.39414	0.04464	-0.15601
8	-0.02656	0.47214	0.06611	0.19587	-0.52623	-0.10959
9	-0.92827	-0.14177	0.22793	0.12145	-0.03293	0.04690
10	-0.91582	-0.09806	0.17165	0.13010	-0.01112	-0.02959
11	-0.12779	0.32384	-0.57183	0.40583	-0.28349	-0.05354
12	-0.91909	-0.05816	-0.03135	-0.06432	-0.01624	-0.00949
13	-0.29466	-0.03564	0.61876	0.13303	-0.05837	-0.17447
14	-0.00139	-0.01613	0.09341	0.63678	-0.07252	0.00514
15	0.35271	0.12899	-0.09743	-0.31107	-0.17037	0.07689
16	0.87776	0.13345	0.02214	0.14100	0.01811	-0.15383
17	-0.21073	0.17531	-0.25151	-0.13040	0.06137	0.71567
18	-0.16384	-0.01677	-0.61067	-0.04093	-0.07605	-0.00698
19	0.30928	0.13667	0.03834	0.45942	0.43333	0.10947
20	0.57902	0.23576	-0.40698	0.47473	-0.08759	0.22514
21	-0.06664	-0.52388	0.15304	-0.02859	0.05466	-0.13029
22	-0.67400	-0.15985	-0.35370	-0.15853	0.15971	0.20682
23	-0.13169	0.08209	0.37711	0.04896	-0.27823	0.06741
24	-0.10534	-0.38164	0.22195	-0.15162	-0.43729	0.50633
25	-0.80106	-0.20397	0.12259	-0.23657	0.11800	0.00151
26	0.30282	0.32641	0.14905	0.13221	-0.06959	0.61388
27	0.08986	-0.02600	-0.11965	-0.21005	-0.12740	0.04733
28	0.61930	-0.04747	-0.00192	-0.03602	0.06058	0.21082
29	-0.77061	-0.16214	-0.27948	0.13025	0.10321	0.01609

Varimax rotation : non-asthmatics

ROTATED FACTOR MATRIX

VARIABLE	FACTOR 1	2	3	4	5	6
1	-0.21228	0.04888	-0.26736	0.17454	0.32953	-0.27194
2	-0.21978	-0.13311	-0.02726	0.16933	0.13581	0.41659
3	0.49915	0.30874	-0.34883	-0.10882	0.01572	0.07276
4	-0.06659	-0.01320	0.12723	0.03033	0.80677	0.05701
5	0.10192	-0.83189	-0.19173	0.07228	0.26222	0.34571
6	-0.10040	0.82230	-0.03131	-0.09760	0.02336	-0.06439
7	0.20926	-0.39191	-0.12343	0.03526	-0.01398	0.42198
8	0.07626	0.48533	-0.09848	0.03275	0.46376	0.06259
9	0.93016	-0.05044	0.11787	-0.14588	0.13441	0.14452
10	0.92095	-0.00765	0.04166	-0.08866	0.07991	0.13748
11	0.19549	0.29288	-0.00300	0.67161	0.16800	0.31633
12	0.94191	0.04195	0.04010	0.09237	0.04132	-0.07268
13	0.25741	0.01445	-0.14988	-0.57199	0.13396	0.12314
14	-0.00892	-0.06818	0.07695	-0.00951	0.12830	0.65516
15	-0.33577	0.11870	0.01325	0.05343	0.12675	-0.34840
16	-0.89962	0.03788	-0.19509	-0.06514	-0.08838	0.11655
17	0.29430	0.18540	0.77314	0.24228	0.07008	-0.02871
18	0.19754	-0.02210	-0.01440	0.62316	-0.02300	-0.07744
19	-0.30170	0.06444	0.19802	-0.04287	-0.38184	0.55215
20	-0.53705	0.11852	0.26287	0.43512	0.06547	0.48490
21	-0.01142	-0.52473	-0.17052	-0.17756	-0.01329	0.00984
22	0.70561	-0.10237	0.24485	0.35926	-0.13078	-0.10700
23	0.13145	0.11133	0.07278	-0.33092	0.35035	0.02567
24	0.08223	-0.37286	0.46140	-0.21442	0.62491	-0.09301
25	0.79584	-0.10144	0.02421	-0.11052	-0.06255	-0.20895
26	-0.24391	0.28560	0.66570	-0.14357	0.22095	0.21032
27	-0.08534	-0.02367	0.00431	0.09730	0.10322	-0.23044
28	-0.63185	-0.11811	0.17729	-0.06052	-0.02312	0.01686
29	0.78828	-0.11114	0.08056	0.33581	-0.09228	0.15310

Oblimin rotation : non-asthmatics

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SOME PSYCHOLOGICAL AND PHYSIOLOGICAL CONSIDERATIONS OF BREATHLESSNESS

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Breathing has been identified with man's feelings since time immemorial. The agony of breathlessness is taken almost for granted, yet the emotional consequences of the symptom have attracted remarkably little scientific examination.

In a psychiatric study of the dying, Hinton (1963) observed that only one-fifth of patients with dyspnoea were relieved by treatment, in contrast to those suffering pain of whom four-fifths were relieved of their distress. There was clear evidence of anxiety in twice as many dying patients who were breathless as compared with those who had pain, nausea and vomiting or malaise.

Campbell and Howell (1963) put forward an attractive theory to account for the neural basis of the sensation of breathlessness: that it reflected an appreciation of length-tension inappropriateness. They remarked how the symptom could arise in anxious patients but they did not refer to activity in neural structures consequent to the unaccustomed afferent signals and appreciation of their unpleasantness. At a symposium on *Breathlessness* in 1965 (Howell and Campbell, 1966) there was almost no mention of the emotional response, at either psychological or physiological levels, which accompanies hindrance to breathing.

Moruzzi and Magoun (1949) discovered that the reticular formation in the brainstem is responsible for the state of wakefulness. Since then neural pathways have been identified whereby signals from the peripheral nervous system are relayed to structures in the limbic system, where the emotional state is modulated (Smythies, 1967). Such structures can be influenced by signals which fail to reach consciousness and also by activity primarily in the cerebral cortex. Thus, information regarding pulmonary length-tension relationships, when inconsistent with the physiological requirements for alveolar ventilation, can not only reach consciousness but can also excite psychological responses, with associated psychophysiological

activity. Severinghaus (1966) pointed out that increased wakefulness from feedback of information on inadequate respiration can benefit associated dyspnoea.

The gamma motor system projects diffusely and, unlike with alpha motor neurone activity, is incapable of precise control. If additional gamma activity causes increased alveolar ventilation, it will simultaneously spread to accessory muscles of respiration and often beyond, causing generalized muscle tenseness. Involuntary increase in muscle activity also develops in hypocapnia, and indeed such change can be detected by electromyography long before the development of clinical tetany.

Emotional arousal probably produces increased gamma activity in respiratory muscles involuntarily, just as it does elsewhere; change in respiration can then occur, with similar consequences to those associated with deliberate overbreathing. Kerr, Dalton and Gliebe (1937) showed that hyperventilation produced tetany easily in patients with anxiety neurosis. Frequently it is impossible to decide in a particular patient whether overbreathing occurred deliberately or otherwise, especially if there is slight pulmonary or cardiac pathology, as well as neurotic disorder. The distinction of malingering from anxiety or hysteria is often involved and, whereas recognition of the aetiology as psychogenic is usually sufficient for appropriate management, psychiatric exploration for the underlying cause, or at least for one contributing to the distress, can be rewarding (for example, Burns and Howell, 1969).

As well as the direct neural activity that results from emotional arousal ACTH will be released through hypothalamic connexions, thus increasing circulating steroid levels. Associated sympathetic stimulation will cause the release of adrenaline and noradrenaline, both of which produce hyperventilation by a direct effect on the brain (Whelan and Young, 1953; Young, 1957). In this way, a complex servosystem operates, involving muscle, neurotransmitters and endocrine metabolism, and through which the response to a threatening situation—such as dyspnoea—may feed back on respiration and cardiac action, as well as on the feeling state. Some of these aspects of the psychophysiology of breathlessness are illustrated in Fig. 1.

The sensation of breathlessness can accompany increased respiratory frequency or volume, both of which can be measured precisely. This sensation can also be due to mechanical hindrance of the act of breathing, the perception of which cannot be measured directly. The degree of asphyxia experienced is a subjective phenomenon, and the observer has to rely on communication from the patient; usually this has to be in a languag

with few suitable quantitative terms, such as mild, moderate or severe. Measurement is made easier if the subject is presented with an analogue scale on which he can indicate his feelings (Aitken, 1969).

Comroe (1966) emphasized that dyspnoea "is subjective . . . it involves both perception of the sensation by the patient and his reaction to the sensation." Unlike many measures related to metabolism, there is wide variation between persons, and between occasions in the same individual, with regard to response to a threatening situation. The nature and degree

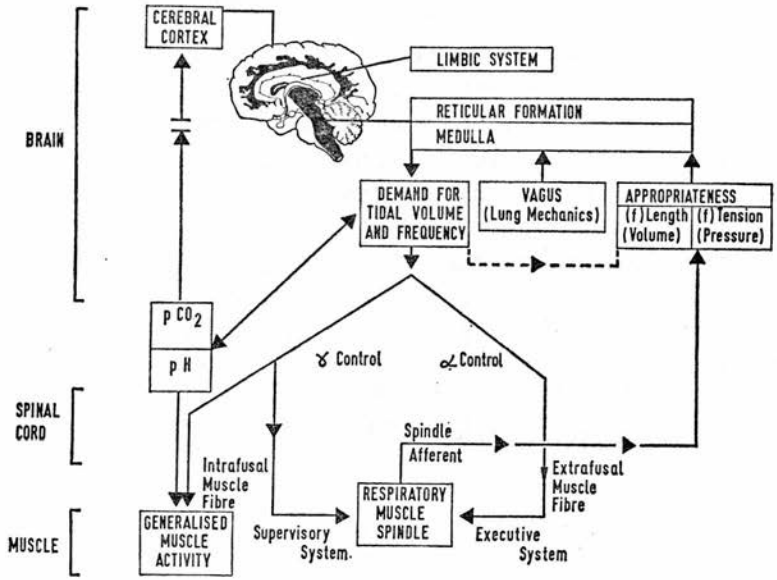

FIG. 1. Some aspects of the psychophysiology of breathlessness.

of the response, both psychic and physical, are dependent upon personality traits and are influenced by previous experience often far remote in time.

Christie (1935) gave detailed accounts of different types of respiration in neurotic patients. Feleky (1916) drew attention to the way in which different emotions influenced the pattern of respiration, and Finesinger (1944) to the effect of thoughts with different emotional implications on this pattern. Mezey and Coppen (1961) showed that anxious patients hyperventilate for unit oxygen extraction and have a raised oxygen consumption, particularly during and after exercise. After studying hyperventilation in normal subjects, Fink (1961) concluded that cerebral activity associated with wakefulness is the main determinant of respiratory drive, and that the partial pressure of carbon dioxide in arterial blood

(Pa_{CO_2}) merely augments the effect of this activity. These studies emphasize that in the investigation of breathlessness results can be misleading unless attention is paid to the emotional state of the subject.

Psychiatrists have made intensive studies of selected patients with bronchial asthma and seldom of those with other pulmonary disorders. Like allergists, they have stalked the aetiology of bronchospasm, only to conclude that multiple factors seem probable in most cases (Rees, 1956). Dyspnoea may be felt as endangering life to a greater extent than almost any other symptom: to the patient, the experience of dyspnoea is regarded as something which, if it worsens to asphyxia, must inevitably be fatal. Having this quality, it must often be associated with anxiety, the lessening of which is likely to mitigate the subjective distress caused by the breathlessness. The issue of interest to us is what determines the extent and quality of such emotional arousal in an individual case of bronchial asthma.

Psychiatrists usually study asthmatic patients referred to them by physicians; and conclusions could have been biased due to their sample being neither representative nor compared with controls studied identically. In order to illustrate our approach to the examination of the consideration under review, we shall now report some results of a study on which we are currently engaged regarding the psychopathology of bronchial asthma. Further details about the methods and preliminary results have been published elsewhere (Aitken, Zealley and Rosenthal, 1969).

PSYCHOPATHOLOGY OF BRONCHIAL ASTHMA

Description of subjects

Twenty-four patients with bronchial asthma were selected at *random* from a register of patients attending a local Asthma Clinic. Sixteen were female, eight were male and their mean age was 40 years (range: 20–56). They had had their first attack of asthma an average of 18 years previously (range: 1–47).

Seventeen patients (71 per cent) had noted precipitation of certain attacks by upper respiratory tract infection, and ten (42 per cent) by contact with some kind of animal or vegetable matter. Twelve (50 per cent) acknowledged that disturbance of emotion precipitated at least some of the attacks.

Each patient was matched for sex, age (±8 years) and social class with either a healthy normal or an anxiety neurotic subject. These two control groups provided bipolarity of neurotic response, with which the asthmatics could be compared.

The asthmatics had a reduced mean forced expiratory volume in the first

second (FEV_1) compared with the normals and neurotics. There were no significant differences between the groups either in forced vital capacity (FVC) recorded at the time of testing or in FVC predicted from age and height nomograms (Needham, Rogan and McDonald, 1954). The means and ranges of these measurements are shown in Table I.

TABLE I

LUNG FUNCTION TESTS

Lung volumes		*Asthmatics* ($N=24$)	*Controls* *Normals* ($N=12$)	*Controls* *Neurotics* ($N=12$)	*Analysis of variance between groups* F	$P<$
FEV_1:	mean	2353	3339	2802		
(ml)	range	570–4050	1850–4530	1400–3680	6·3	0·01
FVC:	mean	3486	4254	3663		
(ml)	range	900–6000	2600–5550	2200–5600	0·9	N.S.
Predicted FVC:*	mean	3396	3476	3180		
(ml)	range	2431–4968	2531–5250	2461–4610	0·4	N.S.

* From nomogram by Needham, Rogan and McDonald (1954); in this and subsequent Tables II–VII) N=number of subjects in each group; N.S.=not significant.

Assessment of personality traits at interview

Two psychiatrists recorded independently the presence of a selection of traits. The proportions of subjects with those traits which discriminated significantly between the groups are shown in Table II. It can be seen that asthmatics were characterized by each trait more often than the normal

TABLE II

A SELECTION OF THE PERSONALITY TRAITS ASSESSED AT INTERVIEW BY TWO RATERS

Personality traits	*Asthmatics* ($N=24$) (*Percentage*)	*Controls* *Normals* ($N=12$) (*Percentage*)	*Controls* *Neurotics* ($N=12$) (*Percentage*)	χ^2	$P<$
Obsessional	54	0	58	11·5	0·01
Lack of confidence	54	8	83	13·8	0·001
Sensitive	58	33	92	8·6	0·02
Anxious	54	33	100	11·9	0·01
Dependent	29	16	83	12·3	0·01
Unstable mood	4	0	50	16·2	(0·001)*

Table II shows the percentage of subjects in each diagnostic group who were assigned the traits sted: a trait was assigned if one or other rater felt it was markedly present, or if both raters considered was present to a moderate extent.

The following traits were found no more commonly in one group than in another: irritable, solitary, ysterical, schizoid, submissive, timid, paranoid, cyclothymic, hostile-aggressive, competitive-ggressive, "bottled-up-feelings".

* This P value is in parentheses because, in calculating χ^2, the "expected" value in three of the six ells was less than five.

controls but less often than the neurotics. Nearly as many asthmatics as neurotics were obsessional while, in contrast, only one was considered unstable in mood. Rees (1956) found these same traits in asthmatics more commonly than in controls after detailed study of over 400 cases.

Psychometric tests of personality

The Taylor Manifest Anxiety Scale (TMAS) (Taylor, 1953), Foulds' Hostility Questionnaire (HDHQ) (Caine, Foulds and Hope, 1967) and the Eysenck Personality Inventory (EPI) (Eysenck and Eysenck, 1964) discriminated significantly between the groups of subjects (Table III). The

TABLE III

PSYCHOMETRIC TESTS OF PERSONALITY

		Controls		*Analysis of variance between groups*	
Personality tests	*Asthmatics* ($N=24$)	*Normals* ($N=12$)	*Neurotics* ($N=12$)	F	$P<$
Eysenck Personality Inventory:					
Neuroticism score	10·3	4·2	15·8	18·2	0·001
Extraversion score	9·6	9·5	10·0	0·1	N.S.
Lie score	3·8	4·8	3·9	1·4	N.S.
Taylor Manifest Anxiety Scale:					
Total score	14·1	5·8	29·0	27·5	0·001
Foulds' Hostility Questionnaire:					
Total score	14·7	8·8	22·5	15·0	0·001
Direction score	+3·8	+0·3	+7·3	3·6	N.S.

Each value is the mean score of the group. Where the F value is significant, t tests were done between pairs of groups and each was found to differ significantly at least at $P < 0·01$.

asthmatics reported more anxiety, neuroticism and hostility than the normal controls, but less than the neurotic controls. As these results were comparable with the observations made at psychiatric interview, we deduce that both assessment techniques are valid for these subjects. On no test did the asthmatics' scores differ significantly from published data on normal people.

Feeling of resistance to breathing

Campbell and his group found that in normal subjects the threshold for detection of resistance to breathing was at an added elastance load of 2·4 $cmH_2O/l/s$ (Campbell *et al.*, 1961*b*) and a resistance load of 0·59 $cmH_2O/l/l$ (Bennett *et al.*, 1962). Aitken (1969) examined the relationship between subjective experience of resistance to breathing and pressure loading on expiration. He found that the relationship was curvilinear, obeying the Weber-Fechner Law as described with other sensory modalities.

We were interested in the question: What degree of asphyxia do our groups of subjects perceive, or communicate that they perceive, when exposed to pressure loading in an external airway?

Subjects exhaled against a selection of pressure loadings between two and cmH_2O in a balanced design. They wore a face-mask (RAF type P) and helmet in which were microphone and earphones. A solenoid-operated open/shut valve was attached to the exhalation tube from the

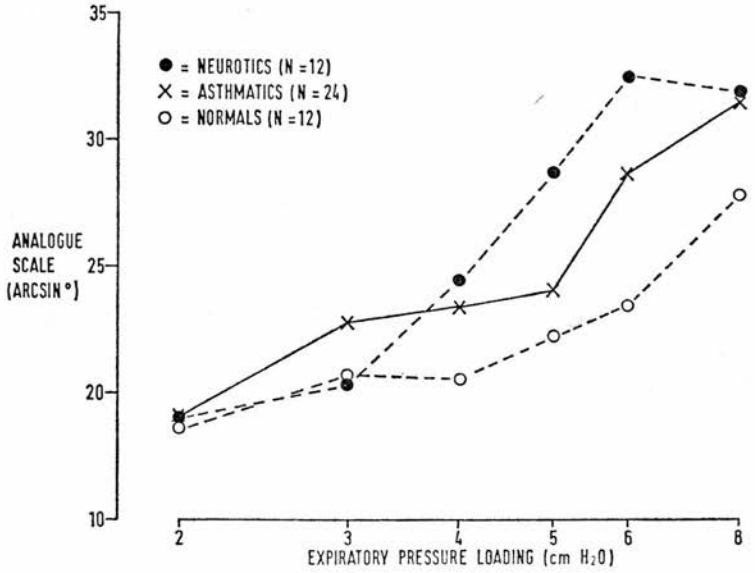

FIG. 2. Feeling of resistance to exhalation at different pressure loadings. The points represent the means obtained on two occasions from each subject in the groups. Measurements in mm from the threshold end were transformed to the *arcsin* to achieve normality of distribution before calculation (Snedecor and Cochran, 1967).

task: when mask pressure exceeded the selected value, the valve opened so that expiration could proceed unimpeded. Subjects communicated their perception of the degree of asphyxia by marking a 10-cm line, the two ends of which were defined as representing "no resistance to breathing" and "impossible to exhale" respectively (Aitken, 1969).

For the different pressure loadings used, the mean scores on the analogue scale for each group are shown on Fig. 2. There were no differences between groups at the lowest value but, as the pressure loading was raised, the neurotics indicated that they experienced a greater degree of asphyxia than the other two groups. Unfortunately the differences on raising the

pressure loading did not become significant even between neurotic and normals ($F=2\cdot53$, $P<0\cdot15$; for $P<0\cdot05$, $F=4\cdot38$). Nevertheless the consistency of the response pattern is striking and, as in the examinatio of personality traits, the asthmatics usually lay intermediate between th two control groups. The probability that such an order between th groups could have occurred by chance on the six occasions is 0·03 (Kendall' coefficient of concordance = 0·58).

Clearly the method used to look at this question cannot distinguish between patient-differences in subjective experience or in the communi cation of perceived sensations; such differentiation demands insight int the individual subject's perceptual and intellectual functioning, and is difficulty inherent in this type of inquiry (Ingham, 1969). Whatever th reason, it seems that asthmatics as a group tended towards the neuroti mode; they communicated appreciation of greater resistance to breathin for a given expiratory pressure loading more than normal controls did.

Examination of cardiopulmonary function

There were no significant differences between the groups in heart an respiratory rates, and minute and tidal volumes. The means and rang between subjects when exhaling against no added pressure loading ar shown in Table IV. For heart and respiratory rates, the means of th asthmatics lay intermediate between the two control groups. For minut and tidal volumes, the asthmatics hyperventilated compared with th

TABLE IV

MEASUREMENTS OF CARDIOPULMONARY FUNCTION AT REST

			Controls		*Analysis of varian between groups*	
Cardiopulmonary measures		*Asthmatics* ($N=24$)	*Normals* ($N=12$)	*Neurotics* ($N=12$)	F	$P<$
Heart rate:	mean	76·2	70·3	77·0	1·31	N.S.
(beats/min)	subject range	57–101	52–92	66–97		
Respiration rate:	mean	14·6	12·6	15·5	1·24	N.S.
(breaths/min)	subject range	6–27	9–16	9–22		
Minute volume:	mean	7·9	6·6	7·3	1·77	N.S.
(l/min)	subject range	4·4–13·0	4·1–8·8	4·8–11·0		
Tidal volume:	mean	0·59	0·54	0·50	0·77	N.S.
(l/breath)	subject range	0·36–1·32	0·42–0·75	0·30–1·09		

The values are based on the means of seven readings in each subject taken at 30-s intervals duri the first 3 min of recording. Heart rate was read from an instantaneous ratemeter, using a suital lead of a standard electrocardiogram as input. Respiration rate was read from another ratemet the input signal to this was taken through a zero switch from a Greer Manometer (type A10 capsu on a pneumotachograph attached to the mask inhalation tube. The inhalation signal was also in grated, and displayed on a pen-recorder for measurement of ventilation volume. (This system h been calibrated for sine wave flow and found to have 95 per cent confidence limits ± 0·6 l/min.)

ıealthy controls. The ranges between subjects were large, inviting peculation on the contributory factors.

When exhaling against pressure loading there were significant changes ı heart and respiratory rates, but there was no consistent trend in the hanges which were observed (Table V). Tidal and minute volumes ıcreased considerably, by an amount that was a function of the pressure ɔading but not of subject group (Fig. 3).

Campbell and co-workers (1961*a*) have reported a similar increase in

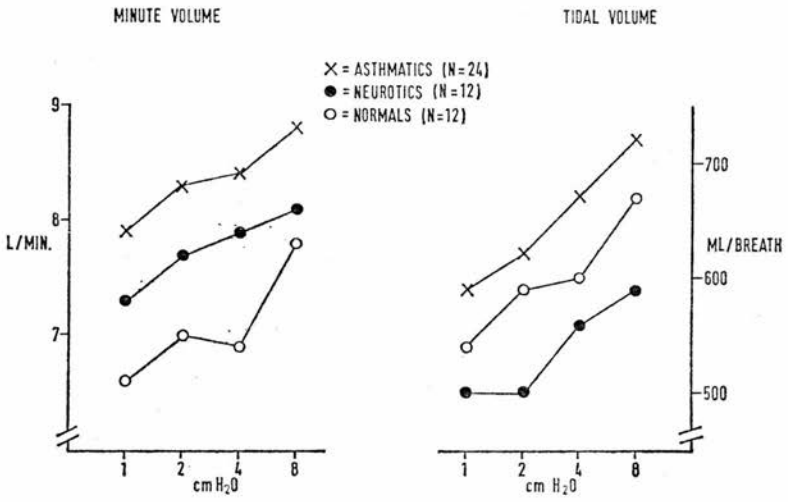

FIG. 3. Ventilation response to pressure loading: (*a*) minute volume; (*b*) tidal volume.
No additional loading was applied for the scores at 10 mm water, this being the pressure needed to operate the internal mask-valve itself.

-lal volume on threshold loading of breathing in conscious healthy -bjects; they noted that on expiratory loading there was an increase in ıd-inspiratory lung volume. Breathing against resistance increases pres--re in bronchial airways and this in turn can dilate bronchioles. A central -tegrative mechanism must be involved to account for the increase in -spiratory volume as a tactic to overcome expiratory pressure loading. our subjects, such a mechanism seemed to operate in a similar way ·espective of whether they were asthmatics, normals or neurotics.

TABLE V

MEASUREMENTS OF CARDIOPULMONARY FUNCTION WHILE EXHALING AGAINST PRESSURE LOADING

Cardiopulmonary measures	Pressure loading (mm water)	Asthmatics (N=24)	Controls: Normals (N=12)	Controls: Neurotics (N=12)	Analysis of variance: Between groups F	Between groups P<	Between pressures F	Between pressures P<
Heart rate: (beats/min)	10	76·2	70·3	77·0				
	20	74·9	68·9	76·8				
	40	74·7	69·9	77·0	1·5	N.S.	3·6	0·05
	80	75·9	71·4	78·8				
Respiration rate: (breaths/min)	10	14·6	12·6	15·5				
	20	15·4	12·7	16·3				
	40	15·1	12·1	15·0	1·4	N.S.	3·1	0·05
	80	14·2	12·3	14·4				
Minute volume: (l/min)	10	7·9	6·6	7·3				
	20	8·3	7·0	7·7				
	40	8·4	6·9	7·9	1·5	N.S.	7·2	0·00
	80	8·8	7·8	8·1				
Tidal volume: (l/breath)	10	0·59	0·54	0·50				
	20	0·62	0·59	0·50				
	40	0·67	0·60	0·56	0·7	N.S.	8·4	0·00
	80	0·72	0·67	0·59				

These figures are the means of six readings in each subject taken at 30-s intervals during 3 m exhaling against the stated pressure loading. Preceding each period subjects breathed for 3 min agai no additional loading.

Mask pressure was measured with a Greer manometer (type A300 capsule); the instrument w modified with limit detectors and relays so it could open the solenoid-operated valve when a select pressure loading was exceeded.

Relationship between severity of asthma and psychometric test scores

There does not seem to be a universal index for severity of the asthmat diathesis. Only one patient (the only chronic bronchitic in the asthn sample) was dyspnoeic during investigation, though ten (43 per cen had an FEV_1 less than 70 per cent of their FVC. Fourteen of the patien (58 per cent) had been in hospital for the treatment of asthma, and fourte were also taking prednisolone regularly with a mean daily dose 6·7 mg.

The asthmatic patients were divided into sub-groups by these criteri and the psychometric test scores examined. The only significant differen was in the TMAS, where the mean score was lower for those taking pre nisolone (11·1) than for those not on this treatment (18·2) ($P<0·05$); th implied less proneness to anxiety in those patients on a regular stero regime, which supports observations by Baraff and Cunningha (1966).

Relationship between measurement of cardiopulmonary function and Taylor Manifest Anxiety Scale scores

The index of psychopathology which discriminated best between the groups was the TMAS score. In order to look at any association between psychopathology and cardiopulmonary function, correlation coefficients were calculated between the TMAS scores and the physiological measurements recorded when breathing at rest (Table VI).

TABLE VI

CORRELATION COEFFICIENTS BETWEEN TMAS SCORES AND MEASUREMENTS OF CARDIOPULMONARY FUNCTION

Cardiopulmonary measures	*Asthmatics* ($N=24$)	*Physically healthy subjects* ($N=24$)	*All subjects* ($N=48$)
Heart rate	0·22	0·42*	0·32*
Respiration rate	0·02	0·49*	0·24
Minute volume	0·04	0·06	0·00
Tidal volume	0·02	−0·34	−0·19

* $P < 0{\cdot}05$.
The values constituting Table IV were taken as the measures of cardiopulmonary function.

In the non-asthmatic subjects, there were associations between TMAS scores and both heart and respiration rates: for heart rates, this remained significant when calculated for all subjects together. The inverse relationship between TMAS scores and tidal volumes nearly reached significance for physically healthy subjects. There appeared to be no association between TMAS scores and minute volumes.

Relationship between tidal volume and other variables

There was no evidence that patients with severer asthma (as assessed by taking prednisolone or having a FEV_1 of less than 70 per cent of their VC) had higher heart or respiration rates or minute or tidal volumes. Thus these criteria of severity did not appear to account for the large variation observed in the measures of cardiopulmonary function.

We have just described how some of the variation in heart and respiration rates, at least in non-asthmatic subjects, could be attributed to varying proneness to anxiety (*viz* TMAS scores); but this index of psychopathology did not account for the variation in ventilation volume, particularly the asthmatics who hyperventilated in comparison with both control groups. The asthmatics therefore lie orthogonal to the neurotic dimension, and their hyperventilation cannot be a function solely of this type of psychopathology.

We then looked for an association between tidal volumes and standard measures of lung volume (Table VII). For the asthmatics we found that tidal volume was a function of lung capacity; but this was *not* the case in the non-asthmatics. We also found that the more recent the onset of asthma the greater was the tidal volume.

TABLE VII

CORRELATION COEFFICIENTS BETWEEN TIDAL VOLUMES AND OTHER VARIABLES

Variables	*Asthmatics* (*N*=24)	*Physically healthy subjects* (*N*=24)	*All subjects* (*N*=48)
Duration of asthma	−0·54*	—	—
Age	−0·38	0·19	−0·08
Height	0·41†	0·03	0·20
Predicted FVC	0·54*	−0·09	0·27
Actual FEV_1	0·46†	−0·13	0·15
Actual FVC	0·43†	0·04	0·25
$\frac{\text{Actual FVC}}{\text{Predicted FVC}}$	0·12	0·19	0·06
$\frac{\text{Actual } FEV_1}{\text{Actual FVC}}$	0·00	−0·37	−0·22

* $P < 0{\cdot}01$; † $P < 0{\cdot}05$.

All figures in the last three columns are correlation coefficients between tidal volume and the variables in the first column.

DISCUSSION

In these results we have reported examination of the psychopathology of bronchial asthma from four quite distinct viewpoints—personality traits revealed at interview, psychometric tests, feelings about breathing against resistance, and cardiopulmonary function; and in identical fashion we have examined matched control groups, bipolar for the dimension of neuroticism under study.

The physiological data were recorded with considerable precision and were direct measures of the activity concerned. On the other hand, the psychological data were only indicants of the psychopathology—feelings of anxiety and of asphyxia are not amenable to measurement, scores on reflecting quite crudely the psychological dimension. (The TMA consists of only fifty questions and cannot be expected to embrace the many manifestations of proneness to anxiety. The psychometric tests used are sufficiently reliable to discriminate groups and thus have validity in this sense; but they are inadequate for drawing conclusions about individuals.)

Though tachycardia, tachypnoea and hyperpnoea are signs of disorder n cardiopulmonary function, they can also be determined by neuro-umoral activity related to emotion. By multivariate analysis it should be uite feasible to calculate how much any psychopathology (at least an mprecise measure of it) and how much any pulmonary pathology con-ributed to the between-subject variance of these measures. Our prelim-nary results suggest that heart and breathing rates may be influenced more y an emotional factor than a pulmonary one; and ventilation volume nore by a pulmonary factor than an emotional one. It seems to be an pen question why asthmatics hyperventilate even when free from ronchospasm, but we do not concede that the cause will be found only n the lung; the relationship between tidal volume and duration—rather nan severity—of the disease makes us still speculate about the influence of igher nervous centres.

Our randomly selected sample of asthmatics did not differ from the ealthy population in amount of neurotic disorder. However, more of them emed to have obsessional, underconfident and sensitive traits in their ersonalities than the normal and neurotic controls, even though their nood stability was within normal limits. The significance of obsessional mptoms is far from clear; this character trait was assigned to those who aid excessive attention to tidiness and orderliness and were pedantic and verconscientious with inconclusive and inflexible ways of thinking and cting (Lewis, 1936). As such people tend to feel insecure, it is easy to rmise the effect of bronchospasm, with the uncertain duration and verity of a developing attack, on their mood. They may well tend to ruminate about the condition, be oversensitive to the onset of minimal yspnoea, and fail to habituate to the initial emotional excitation which companies this onset. These reasons may account for the recurrent eclaration of symptoms in some patients, just as Robinson and Wood 968) showed that neuroticism played a large part in the declaration of naemia.

If anxiety can be reduced and confidence gained by mastery of distress, ne patient should be less disabled by his intermittent dyspnoea even if the tiology of the bronchospasm is elsewhere. Behaviour therapy by syste-natic desensitization aims to do just that (Dudley, Martin and Holmes, 968). Should our reasoning be logical, then a formulation of the psycho-athology in terms of a specific phobia is appropriate. Phobic-obsessional sychiatric patients often experience more distress anticipating a feared tuation in fantasy than in reality. Gelder, Marks and Wolff (1967) owed that, if the personality is otherwise stable and the phobia does not

spread to other situations, systematic desensitization is superior to othe forms of psychotherapy. Moore (1966) has reported promising resul treating asthmatics by this method. In one case currently under treatmer by us we have reason to hope we have induced improvement, since ther has been a gradual rise in FEV_1 accompanied by increased subjective well being over the period of treatment. We are currently working on thi development from phenomenology to therapy.

SUMMARY

The psychophysiology of breathlessness is reviewed. The neural basi for Campbell and Howell's (1963) theory on the appreciation of length tension inappropriateness is extended to include emotional factors a contributing to the sensation of breathlessness and subsequent change i cardiopulmonary function.

A study on the psychopathology of bronchial asthma is reported. A representative sample, with matched normal and neurotic controls, wa examined from four distinct viewpoints—personality traits revealed a interview, psychometric tests, feelings about breathing against resistance and cardiopulmonary function. More of the asthmatics had obsessional underconfident and sensitive traits, but there was no greater prevalence o overt neurotic disorder. Neurotics and asthmatics tended to communicat more feeling of resistance when exhaling against pressure loading tha normals; and they also had a relative tachycardia and tachypnoea Asthmatics tended to hyperventilate compared with the controls, and in a groups ventilation volume increased on pressure loading. Rates of breath ing and heartbeat were related to proneness to anxiety, while volume o breathing was related to lung capacity.

Comroe has emphasized that dyspnoea "involves both perception of th sensation by the patient and his reaction to the sensation." (Comroe 1966). Distress from dyspnoea will be enhanced by the prevailing psycho pathology but should be amenable to treatment aimed at the reductio of anxiety, without the psychopathology being asserted as the cause c bronchospasm.

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DISCUSSION

Dornhorst: The use of analogue scoring for measuring such subjective arameters as dyspnoea is a considerable advance and certainly more eliable than asking the subject to describe what he feels in words.

Wood: I suggest that your results, Dr Aitken, do not confirm the Weber–Fechner Psychophysical Laws (Fechner, 1965) but actually refute them. Weber's Law states that the increase in intensity that must be added to a stimulus to produce a "just noticeable difference" is relative to the initial intensity of the stimulus. Fechner extended this law to include the subjective rating of the intensity of an above-threshold stimulus. He suggested that the magnitude of the sensation produced by such a stimulus could be predicted in terms of units of "just noticeable differences". This is sometimes called the Weber–Fechner Law.

Aitken: I apologize for referring colloquially to the Weber and Fechner Laws, but this was a convenient shorthand way of drawing attention to the curvilinear relationship between threshold and increase in sensation with stimulus magnitude. In 1962, I conducted a small experiment with air crew who exhaled against a series of spring-loaded valves and communicated their feelings on a simple analogue scale (Aitken, 1969). There was a rectilinear relationship between the mean sensation scores and the logarithm of pressure load. Extrapolation of the regression to the point meaning "no detection of resistance" gave, very easily, an estimate of threshold. An analogue scale score is not a measure of perception, but only of the subject's communication about the sensation; the score is produced by components from both parts. Such compounding, of course, applies to measurement of all feelings.

Wood: Treisman (1964) claimed that the extrapolation of such a regression line to zero stimulus intensity showed a curvilinear relationship and the point at 0·25 cmH_2O on your line supports this claim, although one should not really generalize from a single point on such a variable.

My results (unpublished) showing that severity of breathlessness using an analogue scale is a good predictor of internal airways resistance, support yours, Dr Aitken. May I make two points about the results of these studies in relation to the Weber-Fechner Law? Patients with high internal airway resistances have a higher threshold for stimulus detection (greater just noticeable differences) than normal subjects do (Bennett *et al.*, 1962; Wiley and Zechman, 1966). Asthmatics also have high internal resistance. Therefore the magnitude of one particular high external respiratory load will be equivalent to a smaller number of just noticeable differences for the patients (asthmatics) than for the normal subjects. The Weber–Fechner Law would predict from this that the patients should rate this added load as less severe than the normal subjects would. Dr Aitken's results, and my own findings, show the converse. This may be because *patients* have a tendency to exaggerate their symptoms. However, ratings of a hig

espiratory load by normal subjects, before and during the elevation of heir internal airways resistances by cotton dust inhalation, show the ame tendencies: when the internal resistance is high the external load is ated as more difficult.

Dornhorst: These findings are not relevant to the situation in asthma ecause the asthmatic's effort is in inspiration; expiration in asthmatics sually remains passive.

Wood: My studies have so far only been concerned with the inspiratory hase of respiration.

Petit: It is misleading to compare the behaviour of asthmatics and normal ubjects exhaling against external resistances because there are many ifferences between the two groups even before one starts the experiment. he asthmatic is anxious during any test of respiratory function simply ecause he is an asthmatic. A chronic asthmatic, also, has long experience observing his respiratory sensations whereas the normal subject has not. nd the asthmatic at rest may already have a tachycardia if he is taking ronchodilator drugs. I do not entirely agree with Comroe's (1966) omment (quoted by Dr Aitken) that dyspnoea "involves both perception f the sensation by the patient and his reaction to the sensation", although f course we must try to distinguish between the asthmatic's sensation of yspnoea and his distressed reaction to it.

Aitken: It is true that we should make this distinction but it is quite ceptable to describe observed phenomenology, certainly in asthma here there have been so few controlled studies. Caution is always ecessary in suggesting explanations for noted differences, so we must e clear what the term *normal* means. Physiologists often use it to mean without pathology" thus denoting "basal", and we have used it in is sense. In contrast, psychologists often use "normal" to refer to a ran- om sample of the general population, where the distribution of scores ay be far from basal. In order to establish where the asthmatic group as situated we needed two control samples, bipolar toward the tremities of the neurotic dimension.

Dornhorst: You indicated your appreciation of problems connected with ese two different meanings of normal when you showed that the thmatics were on the population norm for neuroticism and the normal ntrols were "supernormal". A possible trap would be to have deduced om this that the asthmatics were abnormal in this respect. It is certainly isleading to identify the sensation of dyspnoea with what can be served by measurement; these two parameters are important but parate. Deformation of the chest wall, for example, is the same whether

it is produced as a response to an external resistance or to an equivalen internal resistance caused by airways obstruction, but the associate sensation will be different in each case.

Wood: I have found (unpublished data) that patients can usefull communicate as separate entities the difficulty and the anxiety experience when inspiring against a high resistance (Comroe's [1966] "perception o and reaction to" sensation). The anxiety is highly correlated with neuro ticism, as measured by the Maudsley Personality Inventory.

Campbell: Will you describe this work in some more detail?

Wood: I used a detection technique based on one described by Bennet and co-workers (1962). Normal subjects and patients with chroni airways obstruction were included. Using techniques discovered recentl in psychophysics, a measure of a subject's ability to detect respiratory load of low intensity was obtained (Swets, Tanner and Birdsall, 1961), un contaminated by either the type of instructions or the motivation of th subject. A measure of the motivation in terms of response bias was als derived. The classical threshold is a combination of these two measure in unknown proportions. My results for detection simply confirme previous workers' findings that normal subjects can detect lower respir tory loads (0·24 $cmH_2O/l/s$) than patients (0·86 $cmH_2O/l/s$). Howeve the results also clearly showed that motivation in patients and norm subjects was different; the former were significantly more willing t admit the presence of the stimulus.

Dornhorst: How do these results fit in with the findings in Dr Guz tracheostomized patient who could appreciate levels of external resistanc when he was breathing through his upper airways but not when h breathed through the tracheostomy tube? This patient must hav detected resistances by means of sensation in his upper airways. The seems to be some discrepancy between these results and yours, Mr Woo although one might have expected that, whatever is happening below t tube, patients could detect changes in load through their mouth and pharyn

Wood: Some of our preliminary results do suggest that intra-or pressure is an important determinant in the detection of external respir tory loads. But resistance, which is intra-oral pressure corrected f flow rate (in my experiment), is a better indication of what the subject detecting. Flow rate is not so important but not entirely dispensabl Dr Guz's results require that flow is detected somewhere in the pharyn which contradicts some work of Professor Campbell's group (Benne *et al.*, 1962).

Campbell: The question is whether patients with chronic airwa

obstruction can detect external ventilatory loads more or less easily than normal subjects. Dr Zechman, didn't you show (Wiley and Zechman, 1966) that patients with chronic bronchitis were less good at detecting external loads than normal subjects?

Zechman: No. We studied one bronchitic subject in detail for comparison with our normal subjects. The bronchitic patient had a pulmonary resistance of 4·8 $cmH_2O/l/s$ and required the addition of a resistance of 1·3 $cmH_2O/l/s$ for detection. The average pulmonary resistance of the normal subjects was 1·9 $cmH_2O/l/s$ and they, on the average, detected an increase of resistance of 0·5 $cmH_2O/l/s$. The proportional change, $\Delta R/R_{total}$ (where ΔR is the change of resistance added externally and R_{total} is the initial pulmonary resistance plus the minimum resistance of the breathing circuit) is about 0·25 in each case. The same relationship was obtained when we experimentally increased the pulmonary resistance of our control subjects by various techniques (for example placing them in a five degree, head-down position). We therefore concluded that a certain proportional change rather than the absolute change is used in the detection process. On this basis, the patient with chronic bronchitis has a threshold for detection which is identical with that of our normal subjects.

Dornhorst: Do you mean by proportional that the absolute level of detection was increased in the patients? You would have to make allowance for their higher internal resistance, of course.

Zechman: That is so.

Dornhorst: So these results support what you found, Dr Guz. Could our patient have been abnormal because his upper airways were usually excluded from his effective respiratory passages?

Guz: I doubt it. Dr Newsom Davis was the first person to show the importance of the upper airways in detecting external loads (Newsom Davis, 1967).

Newsom Davis: I studied a group of patients with incomplete lesions of the cervical cord, predominantly involving the posterior columns, with loss of postural sense in the limbs. These patients had difficulty in detecting inspiratory loads when compared to a group of control subjects. When oral sensation was reduced by an amethocaine lozenge, further impairment of detection occurred in the patients with cord lesions but not in the control subjects. This indicated that the patients, deprived of somatic afferent information from the chest wall, were relying upon oral sensation to detect the inspiratory load, through the transient change in intra-oral pressure which is produced by inspiring against the load.

Guz: All these results show is that loading experiments should not be done at the mouth!

Merton: Could this difficulty be avoided if the subject were put in a skin diving-suit and resistances added to the face-piece outside the suit so that changing the resistances would not give rise to pressure changes on the face? Has the same sort of mouth-piece been used in all these experiments? Another discrepancy might be introduced if a face mask were used in some cases and a mouth-piece in others.

Campbell: Certainly mouth-pieces of different sizes may have been used, and the head held in different positions and so on. But I doubt if your solution would work because clues arise from all sorts of sources as well as the upper airways; for example, the mask may fit badly and move during the experiment. It is difficult to devise a technique with external loading in which clues from all other sources are eliminated because one is detecting such a small load.

Dejours: A certain type of breathing pattern exists for every individual practically specific for life (like a finger-print) and not related to compliance or airways resistance or anything else that I know of. Any individual can be recognized by a graph of his breathing pattern. This has nothing to do with the fit of the mask or other external conditions: it depends on the individual personality. I suggest that exercise is not a particularly useful tool for assessing pulmonary function in respiratory disease because of this. The variation in, say, respiratory frequency among even normal subjects on exercise is so wide—from five to twenty-five breaths per minute at least (Dejours, 1961), so one has difficulty in establishing what is normal.

Cotes: Dr Aitken, your asthmatic patients had increased ventilation minute volumes and tidal volumes, but the relationship of ventilation minute volume to tidal volume was the same as in the normal subject. This is also our experience (Cotes, Johnson and McDonald, this volume, p. 297). By contrast, your neurotically anxious patients with moderately increased ventilation had reduced tidal volumes so were genuinely tachypnoeic; that is, their increase in respiratory rate was out of proportion to the increase in ventilation minute volume. Could you expand your comments on the respiratory sensations experienced by these patients?

Aitken: We have few observations other than the measurements I have already described. The neurotics did not complain of breathlessness or distress when breathing against the high expiratory load, nor did the asthmatics or the normal subjects. Occasionally a subject would describe his sensation after the test, but our experience has been that their remarks

did not help us to understand why the neurotics communicated feelings of more load.

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PSYCHOPATHOLOGY IN BRONCHIAL ASTHMATIC PATIENTS

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Summary. *A tripartite study of randomly selected adult asthmatics is reported, observations being by means of interview and psychometric tests and psychophysiological examination while breathing both freely and against external resistance. Results from normal and neurotic controls are compared.*

At interview, traits of obsessionality, dependency, sensitivity, anxiety and low self-confidence were commoner in asthmatics than normal controls.

In tests of neuroticism, hostility and anxiety, asthmatics consistently lay intermediate between normals and neurotics, though their scores were almost all within the published normal ranges.

Breathing against resistance, only the neurotics failed to hyperventilate: episodic asthmatics hyperventilated the most.

As the patients with the more severe pulmonary disorder (asthma of continuous type) revealed less evidence of neuroticism, it was concluded that psychopathology need not be implicated as the cause of the asthmatic diathesis; it is as likely that the concomitant psychopathology only determines the clinical presentation.

PSYCHOLOGICAL and allergic factors are among those implicated in the aetiology of bronchial asthma. As the cause of the illness remains unknown, psychiatrists and allergists have made statements which not only still require proof, but may even defy it.

Many personality traits found in asthmatics are also found in anxious people without respiratory distress—such as pedantic attention to detail, unresolved anger and emotional lability. Examination of asthmatics specifically referred to psychiatrists or allergists may have accounted for the high prevalence of these traits recorded. As severe dyspnoea will evoke anxiety in anyone, knowledge of the emotional state of a **random** sample of such patients is required, in order to ascertain the contribution of emotion to aetiology. This study was aimed to acquire such knowledge; and, by identical examination of controls, permit statements on the probability of the role of certain personality traits.

METHOD

Selection of subjects

Asthmatics. Two registers were available of all patients attending a local Asthma Clinic. One register consisted of patients with chronic or continuous asthma for which the only effective treatment was the administration of corticosteroids (Walsh & Grant, 1966). The other register contained patients with a less disabling disorder, the majority of whom were not treated with corticosteroids; their condition was acute or only episodic in type.

Twelve patients were chosen at random from each register. Care was taken to obviate bias amongst those selected from the registers either toward or against a possible psychogenic aetiology. In both groups, 8 were female and 4 were male.

Controls. Each patient was matched for sex, age ($\pm$8 years) and social class with either a normal or a neurotic subject. This provided 2 control groups, who were selected so that their age (Table I) and sex composition were identical. The **neurotic** subjects were selected from the current patient register at the Royal Edinburgh Hospital; all had been treated as in-patients within the past year, assigned a diagnosis of neurosis with anxiety a prominent feature, and were still attending as out-patients. The **normal** subjects were obtained by asking acquaintances to nominate healthy contacts of the required age, sex and social class.

Measurements

Clinical interview. For both asthmatic history and personality assessment, as much information as possible was recorded in numerical form. For the latter, two psychiatrists recorded independently the applicability of a selection of traits using a 3-point scale.

Pulmonary function tests. Forced vital capacity (FVC) was measured in all subjects: the value predicted from age and height regressions (Needham *et al.*, 1954) was calculated, and the actual FVC was then expressed as a percentage of this. In addition, the forced expiratory volume in the first second (FEV_1) was recorded, using a McDermott dry spirometer.

Psychophysiological examination. This was conducted in a manner reported in an earlier investigation (Aitken *et al.*, 1969). Subjects breathed through a face-mask; facilities were available for the addition of 2 to 8 cm. water elastance loads to expiration by using a pressure-sensitive solenoid-operated open/shut valve. Results were calculated on the mean of 6 readings obtained at 30 sec. intervals.

Psychometric tests. The Taylor Manifest Anxiety Scale (TMAS) (Taylor, 1953), Foulds Hostility Questionnaire (HDHQ) (Caine *et al.*, 1967), and Eysenck Personality Inventory Form A (EPI) (Eysenck & Eysenck, 1964) were administered in the standard way. Analyses of variance were carried out on the scores; where appropriate, the significance of the differences between groups were ascertained by Student's 't' test.

RESULTS

Clinical interview

Asthmatic history. The patients with continuous asthma had their first attack on average 19 years previously (range: 1-47), the episodic asthmatics on average 16 years previously (range: 1-43). The mean age at onset of the continuous asthmatics was 22 years (range: 3-55), and of the episodic asthmatics 23 years (range: 12-43). Ten of the 12 continuous asthmatics had been in hospital for the treatment of asthma, in contrast to only 4 of the 12 episodic asthmatics. This difference in proportion is significant ($\chi^2 = 4.29$; df = 1; $p < 0.05$). All the continuous asthmatics were taking prednisolone regularly, with a mean daily dosage of 6 mg. (range: 2-20 mg.); 2 of the 12 episodic asthmatics were being treated with prednisolone at the time of testing (4.5 and 8.5 mg. daily).

Most patients had identified a variety of factors which seemed to have precipitated attacks. In 8 (67%) of the continuous asthmatics and 9 (75%) of the episodic asthmatics, upper respiratory tract infection was claimed to play a prominent part. Five (42%) of the continuous asthmatics and 7 (58%) of the episodic asthmatics stated specifically that emotional factors precipitated at least some of the attacks. Three (25%) of the continuous asthmatics acknowledged that they were allergic to some kind of animal or vegetable matter; the proportion was significantly greater in the episodic asthmatics of whom seven (58%) acknowledged this ($\chi^2 = 4.29$; df = 1; $p < 0.05$).

The mean FVC per cent of the continuous asthmatic patients was significantly smaller than that of the episodic asthmatics ($t = 2.29$; df = 22; $p < 0.05$), and all the controls; surprisingly, it was not significantly less than predicted as 'normal' (100%) calculated from the standardised age and height regressions (Table I). The mean FEV_1 per cent was lower in the asthmatics than the controls, though the difference between the continuous asthmatics and the normals alone was significant ($t = 2.40$; df = 22; $p < 0.05$).

No patient was distressed by such bronchospasm as was present during investigation, despite the mean FEV_1 being lower than that accepted as normal from a healthy population. There was no evidence that any of the normals or the neurotics had impaired pulmonary function.

Personality assessment. While none of the continuous asthmatics would have been diagnosed as suffering from psychoneurosis, in the opinion of the raters this diagnosis could have been applied to two of the episodic asthmatics. Further, definite neurotic traits were encountered in more of the asthmatics than the normals, especially among those with the episodic condition: Table II shows the number of subjects who were regarded by the two raters as displaying the traits

Table I. Age and pulmonary function test data.

	Asthmatics				Controls			
	Continuous type		Episodic type		Normal		Neurotic	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Age	41.8	12.5	38.8	10.7	40.9	9.6	38.3	10.5
Vital capacity: percentage of predicted by age and height	97	23	116	15	123	21	118	24
Forced expiratory volume in one second: percentage of actual vital capacity	69	12	70	17	79	7	77	9

N = 12 for each group

Table II. Personality assessment.

	Asthmatics		Controls		Significance of differences in proportions (χ^2 test) P<
	Continuous type	Episodic type	Normal	Neurotic	
Obsessional	4	9	0	7	0.01
Dependent	1	6	2	10	0.001
Irritable	4	3	1	5	—
Sensitive	7	7	4	11	0.05
Anxious	5	8	4	12	0.01
Unstable	1	0	0	6	0.001
Hostile-aggressive	5	2	3	7	—
Competitive-aggressive	4	3	5	2	—
Solitary	3	8	5	5	—
Hysterical	0	1	1	4	—
Schizoid	0	1	2	1	—
Submissive	2	5	4	4	—
Timid	5	6	5	7	—
Paranoid	1	1	0	2	—
Cyclothymic	2	2	0	1	—
'Lack-confidence'	5	8	1	10	0.01
'Bottle-up-feelings'	6	8	6	6	—

Numbers of subjects who were assigned a total score of 2 or more (out of a possible 4) by two raters, on the personality traits listed. N = 12 for each group.

listed (for this purpose, a patient was assigned the trait if one or other rater felt it was decidedly present, or if both raters thought it was manifest to a moderate extent). The significance of the differences in proportion between the groups determined by χ^2 tests is also reported.

The table shows that three-quarters of episodic asthmatics were regarded as obsessional and half of them as dependent personalities. Almost a third of all the asthmatics were irritable and half were sensitive and anxious individuals: but they were no more unstable than the normal controls, nor were aggressive traits any more apparent. While half of the asthmatics felt that they lacked confidence, there was no appreciable difference between the four groups in reporting a tendency to bottle-up or suppress feelings, a trait commonly attributed to asthmatics.

Enquiry into the attitudes adopted by the subjects' parents during childhood revealed no differences between what was described by the asthmatics and normal groups: more than half the neurotics implied that one or both parents had rejected them emotionally.

Psychophysiological examination

When breathing against no resistance, there was no significant difference between the groups in mean heart rate, respiration rate, or ventilation volume (Table III). However, it is noteworthy that the mean heart rate of the neurotics was nearly 8 beats per min. higher than the normals, the respiration rate 4 breaths per min. faster and the ventilation volume 1.6 l. per min. greater. The mean values for the asthmatic patients lay inter-

Table. III. Psychophysiological examination.

	Asthmatics		Controls	
	Continuous type	Episodic type	Normal	Neurotic
Heart rate (beats/min.)	75.9	74.3	69.5	77.1
Respiration rate (breaths/min.)	13.6	15.1	12.4	16.1
Ventilation volume (l./min.)	7.20	7.87	6.08	7.68
Change in physiological values when breathing against resistance (log units):				
Heart rate	—0.007	0.0016	0.0033	0.0021
Significance of t test	NS	NS	NS	NS
Respiration rate	0.0068	0.0017	—0.0143	—0.0207
Significance of t test	NS	NS	NS	NS
Ventilation volume	0.0614	0.0436	0.0750	0.0191
Significance of t test	0.01	0.01	0.01	NS

Each value is the mean obtained from 12 subjects

mediate between the neurotics and the normals, except for the episodic asthmatics whose mean ventilation volume was the highest. The means for the episodic asthmatics lay closer to the neurotics than those for the continuous asthmatics, except for heart rate.

When breathing against resistance, there was no significant alteration in heart or respiration rates. However, ventilation volume increased significantly ($p < 0.01$) in the normal controls and the asthmatics; the increase in the neurotic controls was considerably less, insufficient to be significant. Once again the mean value for the episodic asthmatics lay closer to the neurotics, and for the continuous asthmatics to the normals.

The differential increase in ventilation volume reduced discrimination between neurotics and normals when breathing against resistance; it also revealed that the asthmatics hyperventilated (episodic 8.64 and continuous 8.40 l./min.) relative to both control groups (normals 7.20 and neurotics 7.89 l./min.) when exposed to a respiratory difficulty.

Psychometric tests

All the tests used discriminated well in some way between the groups of subjects (Table IV). The most useful in this respect was the TMAS, where all groups were significantly different from one another (p at least <0.05), the order being (from low to high anxiety) normals, continuous asthmatics, episodic asthmatics and neurotics.

The mean scores of the groups on the neuroticism scale of the EPI were in the same rank order, though discrimination was less good—the normals scored significantly lower than all other groups and the continuous asthmatics than the neurotics. The mean scores on the extraversion and the lie scales of the EPI did not differentiate between groups, a finding in keeping with the result of previous studies (Eysenck & Eysenck, 1964). It is, however, noteworthy that the normals had the highest mean lie score.

Two scores are obtained from the HDHQ—total hostility and direction of hostility (intropunitive minus extrapunitive scores). The mean total hostility scores in each

Table IV. Psychometric test scores.

	Asthmatics		Controls		Analysis of variance:
	Continuous type	Episodic type	Normal	Neurotics	significance of differences between groups P<
Eysenck Personality Inventory:					
Neuroticism score	8.3	12.4	4.2	15.8	0.001
Extraversion score	10.6	8.5	9.5	10.0	NS
Lie score	3.8	3.7	4.8	3.9	NS
Taylor manifest anxiety scale					
Score	10.4	17.7	5.8	29.0	0.001
Foulds hostility questionnaire:					
Total score	14.1	15.3	8.8	22.5	0.001
Direction of hostility	2.8	4.8	0.3	7.6	NS

Each value is the mean score of 12 subjects

group were also in the same rank order as the TMAS and neuroticism scale of the EPI. Except between the two asthmatic groups, the mean scores for all groups were all significantly different from one another. Differences between groups in direction of hostility were not significant; however, the asthmatic scores again lay intermediate between the normals and the neurotics, all groups being intropunitive.

We conclude that the asthmatics reported more anxiety, neuroticism and hostility (Foulds' terminology) than the normal controls, but to lesser degree than the neurotic controls. The episodic asthmatics reported more anxiety than the continuous asthmatics.

DISCUSSION

The central question is how much psychopathology was present in our samples of asthmatic patients. Unfortunately, our normal controls may not have been representative of the general population as their TMAS, neuroticism score on the EPI and total score on the HDHQ were all significantly less than reported from larger random samples (Kelly & Walter, 1968; Eysenck & Eysenck, 1964; Philip, 1968); and their mean lie score was the highest of our 4 groups.

Only the mean EPI neuroticism scale score of the episodic cases was notable as high ($t = 2.40$; $df = 2010$; $p < 0.05$) compared with the general population. We have reported that certain neurotic traits, for instance dependency as judged clinically, were found more commonly in these patients than in the continuous asthmatics. In both groups of asthmatics, traits of obsessionality, sensitivity, anxiety and 'lack-of-confidence' were quite common, exactly as shown by Rees (1956)—traits that could easily lead to failure to adapt to distress, with a tendency to reiteration of complaint.

The psychophysiological results suggest that in this setting psychological factors were playing as large a part in the determination of the amount of ventilation as metabolic requirements. Tachycardia, tachypnoea and hyperpnoea are, of course, signs of disorder in cardiopulmonary function as well as of neurotic manifestations. Nevertheless, should the hyperventilation in the asthmatics have been due to pulmonary pathology alone, a higher value would not have been expected in the episodic asthmatics, but rather in the continuous asthmatics. Clearly if such a response occurs due to anxiety in asthmatics with bronchospasm, dyspnoea will be further aggravated.

Many patients with purely episodic asthma may never attend hospital, since the disability is often mild and transitory. Those who do, however, are likely to be one of two types—either they will have made a greater impression of complaint on their family doctor who then referred them to the specialist in the hope of sharing the 'load', or the episodes of asthma will have been of more than average frequency or severity, thus causing particular concern to the family doctor.

In virtually all tests, the asthmatics occupied a position intermediate between the normals and the neurotics: but whereas the continuous asthmatics had evidence of severer pulmonary disorder—in terms of more likelihood to have been in hospital for treatment, a poorer FVC, and a greater need for long-term corticosteroids—they displayed less neurotic traits at interview, had lower psychophysiological activation, particularly in amount of hyperventilation, and obtained no abnormal scores on psychometric tests. 'Somatisation' of psychopathology (as bronchospasm), referred to by Lopez Ibor (1956) could account for the paradox of less overt neurotic personality in the severer asthmatics (*i.e.* those with continuous disorder), but this explanation would still be heuristic.

Psychopathology need not be implicated as aetiologically relevant in the asthmatic diathesis of these patients. Having made the diagnosis of asthma, the family doctor may refer patients to the specialist not solely because of severity of asthma, but also due to the extent of concomitant psychopathology. This hypothesis is now open to test.

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